This magazine is published to promote sound thought upon and concerning industrial medicine and traumatic surgery. To that end it will contain articles, news items, reports, digests, and other presentations, together with editors' comments. The editorial policy is to encourage frank discussion. On this basis contributions are invited.

Industrial Medicine

Reg. U. S. Pat. Off.

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Occupational Dermatitis

—Clarifying the Distinction between Occupational Dermatitis and Dermatosis, two frequently confused Terms; delving further into the Pathogenesis and Differential Diagnosis of Occupational Dermatitis; and emphasizing a Rationale of Therapy depending on the Interpretation of Clinical Findings as related to the underlying Histopathology—

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At the Industrial Health Congress held in Chicago in January of this year, certain reports were given which were later abstracted in the magazine, Industrial Medicine.*[1] Reading these as a group, we were struck by two noteworthy features. The first was that there would not have been such a Congress, if the Section on Dermatology of the American Medical Association had not introduced a resolution which was acted upon at the Atlantic City session where authority was given the House of Delegates to appoint a Council on Industrial Health.

Why should the Section on Dermatology have taken the initiative in this matter of improving industrial health?

Statistics

The answer is found in the statistics quoted by Royd R. Sayers, Senior Surgeon of the Public Health Service, who, in discussing the present status of disease in industry, pointed out that 85% of the compensation cases for industrial or occupational diseases are those relating to the skin.[2] C. O. Sappington, reviewing the year 1937, acknowledges that dermatitis for that period again led all reportable industrial diseases in the states where such information is available, namely, New York, Connecticut and Ohio, while one large insurance company reports over 1,300 dermatitis claims per year, many without disability, but some running the limit of compensation.[3]

Reports for earlier years show that more than 60% of all occupational diseases were consistently accounted for by dermatitis in the states of New York, Ohio and Massachusetts.[4] In Illinois, during the first year of their Industrial Commission up to October 1, 1937, lead poisoning accounted for 47 cases out of the 220 reported, with dermatitis not far behind with 20.[5] In spite of the laxity of the United States in this respect, we are now able to report that with the recent addition of Delaware, Michigan, Indiana, Pennsylvania and Washington, there are at present laws for the compensation of disability arising from occupational diseases in 21 of the 48 States.

The excellent tables compiled by Dr. Sappington include figures for the period 1928 through 1937 for the State of Ohio, which prove that of 12,931 compensable occupational diseases, 8,730, or 67.5%, were due to dermatitis.[6] In New York State for the year 1937, there were 2,675 disease claims closed, of which 1,564 were due to dermatitis, or 58.6%. In 1938, 1,425 dermatitis claims were closed in the State of New York with 701 awards and 724, or over 50%, denials of claims. Of the 701 cases receiving awards, "soap and water" was listed as the causative agent for 382 cases, or more than half of those accounted for, oil followed with 110 cases, dyes in the fur industry came next with 63 cases, Portland cement with 61 cases, while lime was responsible for 54.[7]

With these statistics before us, it is easy to see

* Read at the March, 1939, meeting of the Association for the Advancement of Industrial Medicine and Surgery.

why the Section on Dermatology should have been so vitally interested in the development of organizational means with which to combat this high percentage of dermatitis due to occupational causes.

History of the Industrial Health Movement

THE second noteworthy feature brought out without exception in all the reports given at the Congress, was the comparative youth of the whole industrial health movement in this country. Dr. Stanley J. Seeger pointed out in his introductory remarks that very little interest was manifested in this study 30 years ago, with few persons realizing the importance of disease prevention in this field. The first attempt at coordination of knowledge was made in 1915 when the Section on Public Health, Preventive and Industrial Medicine conducted a symposium on industrial sanitation. In the same year, a review of the compensation laws was presented to the House of Delegates by the Judicial Council, and the very obvious need for further action was clarified. Dr. Seeger believes that the lag in America is due to the tremendously accelerated advance of industry itself, combined with inadequate training for medical men in their regular school courses.[8]

And yet in England we find a very different picture, perhaps because that country was the home of the industrial revolution. Sir E. Graham Little calls attention to a valuable study made by an Englishman, Dr. Thackrah, as early as 1831, entitled, "A Treatise on the Effects of the Principal Arts, Trades and Professions on Health and Longevity." Since 1896, the Home Office, which is in charge of factory inspection, has enjoyed the services of two great medical examiners: Sir Arthur Whitelegge and Sir Thomas Legge who have directed the efforts of the government towards lowering the incidence of occupational diseases. The work in England has been made immeasurably easier by the largest and most carefully compiled vital statistics of any country in the world, a boast of Sir Little's which it is difficult to refute. The Factory Acts, beginning with that of 1833, have resulted in a tremendous body of accurate information, and the more recent legislation under the Workmen's Compensation Acts has produced an even greater amount of important literature covering this body of knowledge.[9] It is this very lack of detailed material for study that is partially responsible for the problem faced by the physician in America while trying to handle adequately the cases of occupational diseases which come to his attention.

The percentages are peculiarly alike in both countries when dermatitis is made the object of study. In Great Britain, a review covering compensation cases resulting from factories and workshops proved that over 50% came under the heading of dermatitis. Other European countries either do not pay any attention to this important phase of industrial health, or the incidence is much less frequent. Dermatits as a source of disability is mentioned in only six other countries, and in these for extraordinarily limited causes. In Denmark, they recognize wood as a potential irritant; in Sweden, arsenic, mercury, light, heat, x-rays or radium; in Germany, there are three classes of injuries: one from galvanization, one from foreign woods, and another form tar; in Russia, from x-ray and radium, and recently in the Irish Free State. Switzerland specifies certain substances which are recognized as incriminating in consideration of skin injuries.[10]

Definition of Occupational Disease

THE Council on Industrial Health in America immediately recognized the importance of the need of clarification of definitions and terminology. Today, state laws differ on terms and definitions. The simplest expression of what occupational disease means is the explanation that it may be defined as a disease arising out of or in the course of work or occupation.

Legally the definition was fixed under Paragraph 28 of the amended schedule in the New York State Workmen's Compensation Law, after the New York Court of Appeals on January 11, 1938, acted on an appeal from a decision of the State Industrial Board in the case of *Goldberg* v. *Marcy Corporation* and *Maryland Casualty Co.* In this case, Shirley Goldberg, a cashier employed in a motion picture theatre, complained that blotches on, and a general weakness of, her feet had been precipitated by alternate extremes of temperature caused by an electric heater which had to be turned on and off as its only means of regulation, and that because of this weakness, she fell on her way to the physician and broke her ankle. The Court ordered that she be paid her compensation because her disablement was due to "the nature of her employment and was characteristic of and peculiar to the employment in which she was engaged," and further defined the proceedings by upholding the Board's decision that "the injuries sustained by said Shirley Goldberg were accidental injuries and arose out of and in the course of her employment," but they did not base this award on the admittance that her feet were weakened by the lack of proper heating facilities, but rather because she suffered the fracture on the way to the physician, a visit ordered by her employer.

For a full understanding of the definition as upheld by the Court, the meaning of the word "nature" must be clarified. The nature of the work does not mean those conditions brought about by the failure of the employer to furnish a safe place to work, but conditions to which all employees of a class are subject, and which cause the disease as a natural incident of a particular occupation, and attach to that occupation a hazard which distinguishes it from the ordinary occupations and their normal hazards. Thus compensation is restricted to diseases resulting from the ordinary and generally recognized risks incident to a particular employment, and usually from working therein over an extended period. Other Court decisions hold that the occupational disease is that which is "the natural and unavoidable result of the conditions of employment."[11]

Definition of Industrial Disease

THE difference between an occupational disease and an industrial disease is inherent in the word "industry," which would allow compensation for those workers only who are employed in a factory or workshop with other workers. Should this narrower classification be the only one used, Shirley Goldberg's ankle would never have become a bone of contention in a court of law, and the cook who pares her finger along with the carrots would not have any redress.

Definitions of Dermatitis and Dermatosis

THE simplest statement of definition would be that dermatosis is a pathological condition of the skin, without reference to cause, and that dermatitis is an inflammatory condition of the skin due to a contact agent.

Occupational dermatitis must be differentiated from occupational dermatoses since the former has to do specifically with the entire group of contact dermatitis in which the anatomical lesion is an intra-epidermal edema arising out of, or in the course of, work or occupation. The reaction in this type is relatively delayed as compared with the reaction in which the corium is directly concerned, and the noxious substance enters the vascular system directly. In the epidermal form of sensitivity covered by the term dermatitis, the hereditary or familial element is generally absent.

Where the medium of absorption, on the other hand, may not always be the skin, and yet where the skin may be secondarily affected, also in the course of, or arising out of, work or occupation, the term "occupational dermatosis" is employed. A specific example would be where inhalation has become the means of absorption, acting as a carrier of the irritating dust to which the individual is allergic, resulting in what is known as a dermatosis, rather than a dermatitis which requires contact with an irritating agent on the epidermis itself. It must be made clear that the dermatoses may include dermatitis, but the latter does not, in turn, include many diseases which come under the former classification.

Description of Occupational Dermatitis

OCCUPATIONAL dermatitis is of the eczematous variety. It is characterized by an inflamation of the skin in which the epidermis is most conspicuously involved, showing the histologic changes known as spongeosis and clinically, the formation of tiny vesicles. An occupational dermatitis of the acute eczematoid type begins as an erythema followed by papules and vesicles, and upon the breaking of the latter, by oozing and crusting, no matter what irritant is the cause.

Etiology

THE degree to which the skin will react to an outside irritant, depends both on the strength of the causative agent, and the susceptibility of the worker. Compensation laws in Switzerland, Great Britain and the United States have assumed that the handling of certain malevolent substances automatically cause injury, and for this reason they are incorporated into the acts. The New York State Compensation Law lists 27 types of disorders with a brief description of the process involved which might have caused the trouble.[12] The listing of these causes greatly reduces the time and expense involved in litigation, since, at the outset, certain substances are capable, per se, of producing injury without regard for the hypersensitivity of the individual worker. After the 27 specific classifications which include, as the last, dermatitis venenata from any process involving the use of, or direct contact with, acids, alkalies, acids or oil, or with brick, cement, lime, concrete or mortar capable of causing dermatitis venenata. Obviously something with wider scope was required, and Paragraph 28 was recently added which reads: "Any and all occupational diseases resulting from any and all employments enumerated in subdivision one of section three of this chapter," which is the subdivision dealing with occupational diseases.

In addition to this list published by the Industrial Board, we have others which were prepared by physicians. The most complete of these lists, alphabetically arranged for easy consultation, was published by Leonard F. Weber under the title of "External Causes of Dermatitis." It undertakes to cover substances which had been reported up to August 1, 1936. To simplfy the use of the list, Weber added another which lists irritants according to occupations during the course of which these irritants are encountered. The original list runs from page 130 to page 161 in very small type, so that its length gives some suggestion of the enormous possibilities for contracting dermatitis in the course of various occupations.[13]

Louis Schwartz published, in October, 1938, a table illustrating the distribution of 3,136 cases for which the industry or occupation of the affected persons was unknown, according to the specific materials constituting each of the 25 major groups of materials. Another table in the same article[14] covers the incidence and cause of industrial dermatosis based on cases reported to the Division of Industrial Hygiene. The 10 leading chemical causes of occupational dermatitis are shown to be petroleum oils and grease, 14%; alkalies, including cement and concrete, 10%; solvents, 10%; chromic acids and salts, 7%; metals including metal plating, 6%; dyes, 5%; plants, 5%; rubber and its compounds, 3%; paints and varnishes, 3%; synthetic resins, 2.5%. In his summary, Dr. Schwartz points out that it is very difficult to correlate accurately figures from all over the country, but concludes by saying: "Nevertheless, these records combined with the inspections, studies and experiences of the United States Public Health Service, in many of the leading industries lead to the conclusion that the annual incidence of occupational dermatoses in the United States is at least 1% for all industrial workers; that 30% of the cases occur in the metal industries; that the food and domestic industries come next in order .... and that alkalies, petroleum products, solvents, plate metals and metal plating, acids including chromic acid, dyes,

rubber compounds and resins are the leading chemical causes."

While discussing excitants, it would be wrong to limit ourselves to the mention of chemical sensitizens and allergens and primary irritants such as caustics, when both mechanical and external physical agents play an important role. Mechanical causes such as pressure, friction, wounds and abrasions are not in themselves frequent causes of occupational dermatitis, but they often open the skin to the action of chemicals or even bacterial infections. This is particularly true in the fur industry where the stretching pins prick the workers' fingers, and in the citrus fruit industry where the long thorns on the bushes scratch the epidermis and hasten dermatitis due to the action of the oil in the skin of the fruit.[15] Other external agents affecting the health of the skin may be classed as physical causes, such as dry or wet heat causing scalds or burns, cold causing frostbite and chilblains, radium and roentgen rays causing burns and malignant changes in the skin of radiologists, and solar radiation causing sunburn, epithelial proliferation, melanosis and utricarial eruptions on the skin of photosensitive workers in the open air.[16]

The susceptibility of the worker to these outside contacts is usually called a predisposition to the irritating agent. This may be congenital in character, causing an idiosyncrasy, or an acquired sensitization. Allergic reaction is the name under which this phenomenon is discussed. In cases which often become a battle between insurance companies and workers, the claim of occupational disease is met with the argument that the individual's idiosyncrasy is more to blame than the substance involved. Sensitization may depend on such a variety of internal and external factors, that the complexity of the subject as discussed in a court of law is sometimes utterly confusing. Age, sex, conditions of pigmentation, nervous factors, the uncertain equilibrium of the endocrine system which is just beginning to take on its true importance, all must be considered as contributing to the sensitization of the individual. In addition, temperature, ventilation, dampness, fatigue, emotional states, all have an importance, difficult to assess in an individual case, but impossible to disregard.[17]

The normal skin offers a mechanical protection to most poisons applied to its surface. The peculiar properties of keratin explain the protection which the horny layer of the skin, composed of this substance, affords the individual. Keratin, which is soluble in a comparatively weak alkaline solution, is resistant to mineral acids even when these appear in a highly concentrated form. The weak spot in the protection afforded by the integument are the follicular openings of the skin, breaking up as they do, the keratin continuity. Dermatitis caused by oils very often starts its inflammatory process in a follicle. The cutaneous blood vessels are offorded the protection of the sebaceous gland secretions. Fat-dissolving agents are therefore responsible for injuries to the skin, since they remove the natural protection and thus permit absorption into the blood stream of contact poisons.[18]

Conversely, should, for instance, the sweat glands become overactive, the chemical content of the perspiration may assist in breaking down the chemical fumes in the air, causing, by the combination of these salts and acids in varying proportions, a dermatitis which would not have developed if the perspiration had not been present. The importance of this factor has been recognized in the performance of patch tests where the patient's perspiration is usually included in the substance to be tested in order to recreate as nearly as possible the original situation at the time of irritation.

In occupational dermatitis, contrary to other types of contact dermatitis or eczema, antibodies have not been isolated although their reaction has been considered. Sir E. Graham Little calls to our attention that some recent studies have confirmed the influence of certain chemical sensitizers, the presence of which in the blood stream or tissues may, in combination with another cause, produce a reaction not attributable to either factor alone. Porphyrin occurring in the blood, the bowel or the urine has a special effect in precipitating solar dermatitis.

There are certain common diseases of the skin which are prone to render the worker exceptionally sensitive to occupational dermatitis, such as seborrhea, ichthyosis and xeroderma. In the British Army it was discovered that seborrheic cases were far more susceptible to septic infection than unaffected soldiers. The incidence was so marked that the Dermatological Section of the Royal Medical Society suggested that sebhorrhea should be made one of the illnesses to prevent the acceptance of recruits.[19]

The hereditary ichthyotic tendency must also be considered as predisposing the skin to sensitization. In this case it is the dryness and scaling of the skin over the front of the tibiae, and the follicular plugging over the outer surfaces of the upper arms that cause the trouble. Persons who have various skin abnormalities such as tumors, scars, nevi, etc., may be considered as suspects for unusual sensitivity to external contactual irritants.

Symptomatology and Histopathology

IN ORDER to facilitate and make more comprehensible the practical recognition of occupational dermatitis and its differential diagnosis from other cutaneous diseases, it is deemed advisable to describe the types of the eruption as they appear as a result of the histological changes that occur in the skin. Dermatitis should be regarded as a catarrh of the skin having the pathological characteristics of an exudative inflammation, as pointed out so lucidly by Highman.[20]

When the skin is contacted by external substances and changes are produced in it, the epidermal cells are first to react in the form of extracellular and intracellular edema. At the same time, the small blood vessels in the papillary bodies dilate, such changes manifesting themselves clinically as erythema. The process progresses, the epidermal edema becomes more marked, the rete cells swell, enlarge and appear ballooned, while in the

corium, perivascular cellular infiltration and edema increase, resulting in the primary lesion: the papule. With the increased edema, the epidermal cells become spongy and break down to form collections of fluid and cellular detritus resulting in microscopic vesicles which eventually become confluent and produce elevation of the fluid containing serum. While the vesicle is developing, edema continues throughout the various layers of the effected epidermis and is also present in the corium.

A more rapid development of the epidermal cells results in a condition of parakeratosis with scale formation. Crusts are the results of the rupture, tearing and escape of serum and drying of the vesicular contents. Such is the development that occurs in acute dermatitis. Usually the inflammatory condition progresses and the connective tissue in the upper part of the corium swells and becomes more edematous, the perivascular infiltration more marked, so that the skin appears thickened, infiltrated and inelastic. In chronic dermatitis, pigmentation follows as a result of the broken down red blood cells. Lichenification appears when there is acanthosis of the rete pegs and upward projection of the infiltration in the papillary bodies.

As has already been indicated, dermatitis is an exudative inflammation presenting clinical phenomena which represent histopathological changes. It presents a definite clinical entity, whose characteristic symptoms are erythema and congestion, swelling from edema, papule formation and vesicles, followed by weeping and moisture of the serous exudate. Crusts and scales are secondary stages, appearing on the erythematous surface, while the lesions tend to form patches with margins fading gradually into the surrounding skin. In chronic cases, the integument is darker red, thickened, infiltrated and the surface shows exaggerated markings. Even in these cases, there is a tendency of the margins to fade away gradually. At any time new vesicles and moisture may appear.

In occupational dermatitis, the lesions develop at sites of contact with the excitants, particularly on the dorsa of the fingers and hands, while in the more generalized type it occurs on those sites where the irritating agents are brought to the skin in the form of gases and vapors, or through the medium of the fingers touching various parts of the body, or by the absorption through clothing.

The history is most important for the establishment of possible exposure of the skin to an excitant during work, and is also valuable in determining how long a period had intervened between the time of the beginning of the occupation and the appearance of the eruption. The past history of the patient should also be thoroughly investigated to discover whether the patient had a previous eruption or whether evidence of an eruption had been present at the time the work was undertaken. It is well to check on any previous treatment, with particular notice as to whether he had visited a dispensary for his skin condition.

The course of dermatitis varies, but the typical cases usually begin with itching or slight burning and erythema, which rapidly becomes edematous and covered with papules and vesicles which dry rapidly and rupture to form crusts. At this time if the patient is protected against further irritation, the course may be shortened. This is very important in establishing the degree of disability and amount of time lost from the patient's work. However, if proper treatment is not immediately instituted and the patient is further exposed to the irritating agent (having been allowed to return to work before the acute condition has been cured) the inflammatory reaction takes on the characteristics of the chronic type. Such a process, once instituted, may run a prolonged course of months or even years, with periods of improvement and exacerbations, with the formation of fresh vesicular lesions accompanied by moisture and crusting, and the eventual development of thick, infiltrated, lichenified and pigmented areas. Throughout the whole course of dermatitis, there is more or less itching. In the beginning it may be mild in nature or show a burning sensation, and later it might be intermittent or provoked by internal or external agents, but in the chronic type, it may be intense and almost constantly present.

In excluding other dermatoses, the symptoms and signs of other conditions must be borne in mind, the relationship of these conditions to constitutional disorders must be considered, and the histopathological examinations of tissue are frequently desirable. The employment of the laboratory for the performance of blood counts, the determination of serum reactions and microscopic examinations of fungi is necessary. And obviously, the patch test should be employed wherever possible.

Patch Tests: Their History

OVER 40 years ago Jadassohn presented the principle of the patch test and applied it to industrial medicine in Germany with wide experimentation, but in the United States and other countries the study has been confined to the last 10 years. Since A. J. Markley reported its use in December, 1928, there have been 51 papers in American medical literature, 29 in French, 12 in German, 11 in British, all the way down to one in Russian, which have definite references to patch testing.[21] The evaluation of the importance of this aid to diagnosis is demanded with accurated differential diagnoses in the settlement of claims. More and more articles appear discussing both the technique and relative importance of these tests.

Technique

IT IS now common knowledge that hypersensitivity may be further tested by renewed contact with the suspected agent, and that such tests applied to the skin of the worker, are known as patch tests, and all studies of contact irritants today include sections devoted to the results of these tests as applies to the sufferers under discussion. As Goldsmith points out,[22] in purely epidermal

sensitization, as in occupational dermatitis, the allergen gives a positive reaction when applied to the skin of a susceptible worker, while intracutaneous tests prove to be negative. Clinically, the seat of the original aggravation might have been either the dermis or epidermis, since one cannot be damaged primarily without secondarily affecting the other.

The physician who applies the test can place no faith in the results if he is not carefully trained in technique. He must learn the proper concentration of the chemical to be used, recognizing in advance just how strong a concentrate may be employed on the normal skin, and for what length of time it can remain there without causing injury or burn possibilities. Goldsmith publishes Mayer's[23] list of correct strengths for testing purposes of a large number of common substances, which, when placed on the unbroken skin of a normal individual for 24 hours, cause no reaction to take place. Another shorter list of proper proportions of substances to be used in patch testing appeared in a study by Goodman and Sulzberger,[24] while E. J. Trow gave extensive tables which he compiled giving dilutions of most test substances irritating the skin in its normal condition, pointing out that the dilutions used in the tests themselves should therefore be made weaker than that point.[25]

It has been shown that differing parts of the body of the same person, differ in degrees of sensitivity, so that the selection of the part of the body to be tested is very nearly as important as the substance to be used in the test. Areas of the skin approaching as nearly as possible to the original site of the inflammation, are usually the best places on which to perform the tests, though it must be kept in mind that this section of the body might possibly have been further sensitized by the presence of the dermatitis itself.

Dr. Schwartz points out[26] that the worker who alone of all his fellows is suffering from a dermatitis, should be patched with the materials used in the course of his occupation. If he is patched with only one substance, then a control patch should be placed upon him. If he is patched with more than one substance, then any negative reaction from one of these substances will serve as a control. When testing solids, the perspiration of the patient obtained from the axilla should moisten the substance. In order to perform more exact patch tests, it will be necessary to have further studies performed by research workers on the chemical contents of perspiration under differing conditions, because it has been noted that the sweat of an individual may vary from an alkaline to an acid reaction, and under the present hit or miss system, we have no idea as to what type perspiration the patient was forming at the time of the original irritation. Sometimes it is necessary before obtaining a reaction, to try several different perspirations of varying hydrogen content.

In addition to the use of dilutions proven to be harmless to the normal skin, the insulating material inserted between the chemical and the adhesive plaster which affixes it, should be a non-irritating substance which could not possibly assist at the reaction. Sawdust, certain rubber compounds, as well as waterproofed cellophane have characteristics which of themselves are capable of causing irritations. Unvarnished cellulose or a thin sheet of mica have been proved most innocuous.

The appraising of the patch test is the most difficult part, since it can only be considered as a corroborative link in the chain of evidence, and must be read with a full knowledge of the particular occupation involved, the history of the dermatitis, and the site and morphology of the lesion. The correct etiology can only be arrived at by these all inclusive considerations.

Positive Patch Tests

SHOULD the patch test give a positive reaction, that merely means that the portion of the skin patched was sensitive to the contact agent at the time of testing. Before we can come to any conclusions that the substance tested was the one responsible for the occupational dermatitis, we must be positive that the patient was exposed to the material in the course of his work in approximately the same strength which has been employed in the test. We must also presuppose that the patient's skin was also sensitive and predisposed to irritation at the time of the occupational exposure, and that his general physical condition was such as to play a contributory role to the founding of his allergy.

When, however, the patch test is positive, and the differential diagnosis also points to a contact dermatitis, and the physical conditions of the occupation involved account for the presence of the substance used in the test, it seems reasonable to quote the findings of the patch test as one part of the entire diagnosis.

Negative Patch Tests

IF THE results of the tests are negative, it is equally inaccurate to conclude that the agent tested had nothing to do with the causing of the dermatitis, since the area tested might not have been sensitive to the contact agent, while the area of the eruption might very well have been. On the other hand, should it be possible to perform the test at the exact site of the eruption after the latter has been cleared of the inflammation, there is the possibility that the skin of that area has been immunized by the diseased condition itself. A negative test might also result from the impossibility of reproducing the exact conditions of the original irritation, since there are certain external conditions almost impossible to repeat, such as climatic, frictional, etc., even if the physician has succeeded in testing with the correct concentration which in itself is a great problem.

In the last analysis, should the tests remain negative, the possibility occurs that the correct substance has not been located with which to test the worker's sensitivity, and having exhausted the chemicals used during occupation, it is wise to test those found at the home of the worker, particu-

larly anything new with which he has made recent contact.

It must be remembered that the reaction to patch testing is not always an immediate one; the time element may vary from a few hours to a few days. While some dermatologists believe that the delayed reaction is due to the fact that the skin was sensitized directly by the patch put upon it, we hold that it merely denotes hypersensitivity. As a further precaution to check on the accuracy of positive tests, enough substances should be tested to discover whether the worker has developed a polyvalent sensitivity, and if this proves to be the case, the patch test loses its diagnostic value.

Differential Diagnosis

FROM the preceding discussion, it should be readily appreciated that any disease capable of producing all the far-flung and variegated manifestations observed in occupational dermatitis, must be carefully differentiated from other clinical disease pictures, particularly since the signs and symptoms simulate those of other dermatoses. To facilitate the distinction of the individual clinical entities, we shall discuss the following conditions:

1. Dermatophytosis and phytids as well as other mycotic processes;
2. Neurodermatitis;
3. Idiopathic eczema of internal origin;
4. Psoriasis;
5. Impetigo;
6. Lichen planus;
7. Scabies;
8. Seborrheic dermatitis;
9. Erysipelas;
10. Syphilis;
11. Parapsoriasis;
12. Mycosis fungoides, and
13. Dermatitis factitia.

Dermatophytosis and Phytids

SOME vegetable parasitic infections, as for example, tinea capitis with its small or large, circular and circumscribed patches covered with whitish gray scales and broken off hairs is not likely to be confused with occupational dermatitis. Similarly, trichophytosis of the body, with its brownish-red circular patches and raised vesicular borders, is not apt to give rise to difficulty. However, dermatophytosis of the hands and feet closely resembles occupational dermatitis. Different investigators, notably Whitfield[27], have variously classified the manifestations of this type into three groups:

1. Acute vesiculo-bullous, with a sudden onset, grouped lesions, markedly pruritic and most frequently appearing with warm weather.
2. Chronic intertriginous with a whitened sodden mass of epithelium between the toes.
3. Chronic hyperkeratotic.

In this latter type, the horny layer has given rise to large irregular masses usually on the plantar surfaces of the feet. The eruption on the hands may be of any type but is usually either vesicular or dry and scaly. The diagnosis can usually be made rapidly by the clinical appearance or by demonstration of the spores and mycelia in the vesicles or scales. Care in microscopy should be emphasized inasmuch as the fungus is usually situated in the roof of the vesicle and easily lost following puncture.

Other points of importance include the history of a dermatitis which existed prior to the work engaged in, as well as examination to determine whether the inflammation is confined to those sites characteristically involved by the invading fungi. The physician should always examine the toes, soles, groins, axillae and anal regions when consulted by patients with dermatitis of the hands. It is of extreme importance to note that while cultures and scrapings are of marked value in diagnosis, many cases of definite clinical dermatophytosis may not be supported by laboratory proof. Many cases of epidermatophytids have been incorrectly called occupational dermatitis by untrained observers. These eruptions occur in association with parasitic infection of the feet, presumably in the nature of an allergic reaction.

Dermatophytosis of the hands is uncommon, but epidermatophytids are of frequent occurrence. They have many distinguishing characteristics. The ids form patches with sharply defined, elevated borders unlike those of dermatitis in that the borders of the latter fade gradually into the surrounding skin. Moreover the central parts of the ids show a tendency towards involution, while in occupational dermatitis the entire patch is of similar appearance. The vesicles which appear in dermatophytids are deeply seated and form tense, thick-walled lesions which, when torn, give rise to pin-head to grape-seed sized dry erosions with sharply defined, undermined epidermal borders, tending to be discrete. The vesicles in dermatitis are more superficial, appear inflamed, are bright red in color, have thin walls and when ruptured or torn, exude a serous fluid to form crusts. The vesicles tend to run together to form patches with no definite border and usually occur on the backs of the hands and fingers in contradistinction to the palmar and intertriginous sites of the ids.

A misconception exists that the fungi are present in the fingers and hand lesions, which is as a rule false, and only rarely can the micro-organisms be demonstrated. The phenomenon depends upon the fact that the fungi are transported to the hands from the feet by way of the blood stream and are almost immediately destroyed by the immunobiologic reaction of the cutaneous tissues. The fungi are occasionally demonstrable by microscopic examination but culture of tissue may be necessary in doubtful cases and may require several weeks for growth. These episodes of id formation usually, but not necessarily, occur in connection with acute attacks of trichophytosis of the feet.

Ids tend to fade with treatment of the feet alone. Dermatitis of occupational origin disappears when the patient stays away from work or is protected from local irritation. The ids are unaffected by

the continuance of work or working conditions, excepting when irritation causes a secondary dermatitis. A great deal of discussion has centered about the value of the trichophytin test, which merely indicates that the patient has or has had a fungus infection and, in this manner, is somewhat comparable to the tuberculin test. It does not indicate the nature, site or intensity of the infection. If negative, the patient may still have a fungus infection, but his resistance and reactivity are so poor that he cannot produce sufficient immunological bodies to give rise to a positive reaction, or the specific guilty fungi may not have been used in the test. If fungi are not found and the clinical picture is not that of dermatophytosis and the patient does not have a positive trichophytin test, then we can definitely state that there is no tinea infection present.

If the diagnosis is made clinically and the test is negative, the clinician must give a poor prognosis in regard to the duration and cure of the lesions.

In the final anlysis, the physician must depend more on the history, the clinical picture of the lesions, the course and the effect of removal from the cutaneous hazards of the occupation and on the clinical acumen of the dermatologist. Fungi occasionally may be found in blood culture during dermatophytosis. Recurrent instances of inflammatory hand eruptions without repeated exposure to previous hazards and the detection of dermatophytosis of the feet, indicate the presence of dermatophytids of the hands and should not be ruled as evidence or recurrence of the previous occupational dermatitis.

Neurodermatitis or Lichen Simplex Chronicus (Vidal)

THIS is generally regarded as an atopic eczema and is easily confused with chronic dermatitis with lichenification secondary to the scratching, prolonged irritation and inflammation. In the differential diagnosis, one of the most important points rests with the elicitation of an atopic family history, the occurrence of infantile or childhood eczema, hay fever, urticaria, asthma, migraine, angioneurotic edema and other vasomotor disturbances, as well as polyvalent reactions to foods, drugs and inhalants. The sites of predilection include the nape of the neck, the popliteal and cubital areas, the extensor aspects of the forearms, the eyelids, the inner and upper parts of the thighs and genitals. In occupational dermatitis, the original as well as subsequent localized lesions almost always involve the distal parts of the upper extremities and especially the dorsa of the fingers, hands and wrists. In neurodermatitis, the lesions are dry and only show moisture in parts that are secondarily irritated, whereas in the occupational dermatitis, the moisture is that of a serous, exudative inflammation with crusting apparent from the onset.

The presence of pruritus in an intense form, is noted in neurodermatitis, and may seriously interfere with sleep. In addition, it is more prevalent in females. The patches of atopic eczema are dry, sharply outlined, and show no relation whatever to the patient's occupation. Patch tests are negative, but scratch and intradermal tests may be positive.

Idiopathic Eczema of Internal Origin

FORMERLY the scrap-basket of dermatological diagnosis, idiopathic eczema of internal origin is rapidly decreasing in its scope and is diagnosed with less frequency. It does, however, offer a problem in differential diagnosis. Essentially a catarrh of the skin due to an internal imbalance, its histology corresponds with that of occupational dermatitis, but in the latter there is a history of exposure during the course of occupation and the occurrence of lesions on sites of contact. In dermatitis of external origin, the vesicles are more superficial, brighter red in color and increase in number as long as the casual agent operates. The lesions do not reoccur when the irritant is removed and a reasonable time allowed for healing. In eczema of internal origin, recurrences are independent of occupational exposures, and relapses occur unrelated to any exciting external factors. Patch tests are positive in occupational dermatitis whereas they are negative in eczema which may, in addition, be accompanied by positive scratch and intradermal reactions to foods, pollen and other substances, usually protein.

Psoriasis

THE typical case of psoriasis occurring on the usual sites of predilection offers but little difficulty in differential diagnosis. It may, however, simulate squamous dermatitis, especially when it involves the hands. The primary lesions of psoriasis are dry papules. These enlarge and become confluent to form dry patches covered with silvery-white, shiny scales. Upon removal of the scales, pin point bleeding points appear. The patches may be present for years and are usually confined to the front of the knees, the backs of the elbows, the scalp and the trunk. The nails are frequently involved and show changes, particularly in the form of stippling. Pruritus is rarely present in psoriasis.

In dermatitis the primary lesions is a vesicle, the lesions form patches with ill-defined borders fading into the surrounding skin and the scales are finer, grayish or yellowish-white in color, not laminated and disclose moisture, and no bleeding points of the underlying tissue when removed. The rare occurrence of vesicular or pustular psoriasis is always accompanied by the characteristic signs, even in doubtful patches, and may be confirmed by the finding of typical efflorescences on sites where occupational dermatitis is rare, as on the scalp and penis. Microscopic study of biopsy material may be necessary to confirm a clinical diagnosis.

Impetigo

THIS disorder is not commonly confused with occupational dermatitis. However, where scratching and uncleanliness have produced a sec-

ondary infection of the dermatitis, difficulty may arise in establishing the relationship of the condition to the occupation. The usual "stuck-on" crusts, location on the face, and history of contagion simplify the diagnosis. But with infection, the dermatitis may lose its exudative or keratinized nature and be transformed into a crusted impetiginous patch. As in all cases, the history is important. The early appearance of the lesion, the duration of the weeping which is only transitory in impetigo, the yellow crust, the presence of impetigo in other members of the family, the essentially bullous nature of the impetiginous lesion and the absence of surrounding inflammation are all aids in differentiation.

Lichen Planus

OCCASIONALLY confusion may result in differentiating chronic occupational dermatitis from lichen planus, notwithstanding the fact that the characteristics of both conditions are usually so distinct. The primary lesion of lichen planus is a red papule which tends to become violaceous, is polyhedral in shape, angular-based and flat-topped with umbilication at times, and is covered with a firmly adherent waxy scale. It occurs on certain favorite sites such as the front of the wrists, around the ankles, on the glans penis and mucous membranes of the mouth. It should be easily distinguished from the vesicular and vesiculopapular eruption of dermatitis with its softer, rounded, crusted lesions appearing on the backs of hands and fingers and becoming moist on slight irritation. The patches that are formed in lichen planus tend to be more sharply circumscribed and traversed by grayish striae. Additional aids in differentiation are the characteristic violaceous hue, the more intense pruritus, the tendency to pigmentation, and the characteristic histology of lichen planus.

Scabies

THIS pruritic disease may present an appearance similar to that of a dermatitis in a pustular or vesicular stage. Usually, the presence of burrows, the noctural itching, the similar affliction of members of the family, the sites of localization on the webs of the fingers, wrists, axillae, breasts, buttocks and penis, together with the microscopic demonstration of acari, suffice to make the diagnosis. In addition, the lesions of occupational dermatitis are more apt to form in patches rather than show discrete vesicular or pustular arrangements.

Seborrheic Dermatitis

IN BRIEF, this condition is differentiated by its predilection for the scalp, the middle one-third of the face, the scapular region, armpits and the trunk, the oiliness of the scalp and face, the reddish-yellow scales and sharply marginated centrally involuted circular patches.

On the extremities, its yellowish scaly nature is typical and rarely, if ever, does it involve the palms or soles.

Erysipelas

ERYSIPELAS may resemble a sharply limited dermatitis. For purposes of differentiation, we keep in mind its brilliant red color, sharply raised borders, glazed surface, marked swelling and constitutional symptoms of fever, malaise and blood response. There is usually no fever with dermatitis, the patches are not sharply circumscribed, there is no induration and more tendency toward vesiculation, while pruritus is more evident.

Syphilis

SQUAMOUS palmar syphiloderma of the tertiary stage may closely resemble chronic squamous dermatitis. In such cases the unilateral distribution, the infiltration and absence of itching are suggestive of lues. A positive clinical diagnosis is often impossible and one must then have recourse to the serological reaction and antiluetic therapy to fix the diagnosis, as well as biopsy.

Parapsoriasis

PARAPSORIASIS in plaques may be differentiated from dermatitis by the presence of superficial, persistent patches that remain dry and resist treatment. The slow, gradual evolution of the patches, the absence of known etiology and subjective symptoms aid in identifying the condition.

Mycosis Fungoides

RECENTLY a case was observed in which the early phase of mycosis fungoides had been diagnosed as an occupational dermatitis. This condition may be suspected when the apparently benign dermatitis tends to persist, becomes infiltrated, develops patches and plaques of various shapes with sharp, elevated borders and is intensely pruritic. In generalized cases, the erythroderma may disclose isolated normal appearing skin areas. Many cases of mycosis fungoides begin with an apparently harmless dermatitis and eventually, in their chronic course, exhibit the characteristic pathology.

In doubtful cases, a general examination may reveal tumors.

Dermatitis Factitia

THIS paper would not be complete without including a short description of dermatitis factitia. The eruptions included under this term are often self inflicted and are feigned eruptions or cutaneous mutilations provoked by the uses of various caustics, alkalies, acids and other irritating agents. They are encountered most frequently in neurotic and hysterical individuals, but they also may be produced to excite interest or sympathy or with the intent to collect insurance and avoid duty.

The lesions vary from mild erythema to severe and extensive ulceration and deformities.

The diagnosis depends upon the absence of definitely recognized lesions of a distinct clinical entity which occur upon areas that are outside the range of the patient's hands. The lesions look arti-

ficial and frequently their borders are sharply defined, clear cut, pointed, angular, band-like and bizarre in appearance. Dots or streaks may be present where the chemical has been applied and where it ran along the surface of the skin. The eruptions are asymmetrical, unilateral and often show relapses during the night unpreceded by any local disturbance because healing is often prolonged intentionally by the patient.

This condition should be differentiated from neurotic excoriations which is also a condition in which the patient produces skin changes but the etiology differs from that in dermatitis factitia in that the patient does not know that he is producing the lesions. The skin condition is produced unconsciously in most cases by the patient absent mindedly removing what he considers to be blemishes or irregularities in the skin. For instance, the picking of pimples or the pulling out of hair from the face and scalp often occur without intent to malinger.

The report of the New York State Department of Labor indicated that in 1938, there were 1,425 cases applying for compensation because of occupational dermatitis. More than 50% of these were not awarded compensation. In private practice it was found that far more than 50% of the patients complain of skin eruptions which in their opinion are the result of exposures in the course of their work, but which prove to be definitely not occupational dermatitis. Among such conditions, besides the diseases already enumerated and differentiated, there are instances of dermatitis venenata from plant contact, dermatitis as a result of irritation by chemicals in homes and environment, urticaria, erythema multiforme, drug eruptions, rosacea, various parasitic conditions, including tinea versicolor, tinea cruris, moniliasis, monilids, lupus erythematosus, erythema nodosum and pityriasis rosea.

In conclusion we wish to emphasize the fact that in the diagnosis of occupational dermatitis, the most important single factor is the recognition of the clinical manifestations of the complaint. There is, however, no one factor upon which a positive diagnosis can be made in all of the cases that are observed. Several facts should be carefully weighed, including the history of previous eruptions, the onset and development of the present complaint, the site of the eruption and relationship of accessory agents as contributing physical and chemical irritants. Of great importance also is the effect of treatment and the changes observed in the eruption when the patient is removed from his occupation, together with the proper evaluation of patch tests.

In chronic and generalized cases, greater difficulty may be encountered than in the more localized instances of occupational dermatitis because of the presence of polysensitivity. Whenever indicated, although this is frequently expensive and unnecessary, the laboratory should be employed as an aid in differential diagnosis and in extreme cases, hospitalization is advised for further observation and accurate treatment.

Treatment

THE treatment of occupational dermatitis should be directed along three lines: namely, preventive, general and local therapy.

Preventive Therapy

IN ANY study of contact dermatitis, the problem of preventing the occurrence of the cutaneaous condition is too often overlooked. The simple routine of frequent bathing by the worker as well as regular laundering of his clothing would often abort many a troublesome dermatitis, therefore much depends on the availability of showers, wash basins in sufficient numbers, good bland soaps, preventive oils such as cotton seed and olive oils and emollients.

Dr. Schwartz suggests[28] that two lockers should be provided for each worker, one for his street clothes, and one for his work clothes, and furthermore that these lockers should be in separate rooms with shower baths between to minimize exposure possibilities. In many types of occupation, the use of gloves for the finer manipulations and closer contacts is advisable. Of more recent development, is the so-called invisible skin for the hands and other parts of the body to protect them against dirt and all substances which might prove sources of irritation. These newer preparations are essentially mixtures of sodium stearate, sodium silicate, glycerin, water and starch, and may be quickly applied to any part of the body. They form a light, thin film, protective for at least eight hours of continued subjection to irritants, and are easily removed by merely washing with water.

An important advance in preventive medicine lies in the selection of the worker himself. Applicants for positions in which there is continuous contact with possible troublesome agents should be carefully examined for the presence of any existing skin or allergic condition. The history or presence of pruritus, eczema, plant dermatitis, ringworm, xeroderma, folliculitis, or similar states should be carefully noted for future reference, and hay fever, angineurotic edema, asthma, urticaria and other allergic states should be carefully considered. If these conditions appear to be extensive, the applicant should be rejected. The subsequent development of allied hypersensitive reactions in this type of individual is all too frequent. The application of patch testing with materials to which the worker will be exposed, before he is hired, is especially important for the allergic or atopic individual.

Little has been said about the machinery per se, as this subject falls more aptly into the province of hygiene and preventive medicine. Of course, the panacea sought for is the seclusion of irritants from contact with employees. Old machines which blow off fumes, smoke, vapors and the like should be replaced with new ones equipped with hooded enclosures. Forced ventilation, enclosed pipes, suction exhausts and other preventive measures within the province of sanitation engineers may be required. As a final check-up, there should be a

systematic inspection to ensure the continued enforcement of the approved standards, both mechanical as regards the factory, and physical, as regards each worker.

The final phase of the preventive aspect of occupational dermatitis has to deal with the education of the worker himself. It is gratifying to note how greatly the incidence of cutaneous conditions may be reduced following some simple, educational health program. The pointing out of health hazards, simple but graphic talks on daily care and sanitary measures, the printing and posting of a code of safety regulations with penalties for their violation, are all helpful in the reduction of cutaneous outbreaks.

General Therapy

AUTHORS too frequently dismiss the problem of internal treatment with the simple and sweeping statement that general therapy is not required. But very often, these simple cutaneous conditions will not respond to local therapy until some slight modification in the general body response is made. It is often surprising to notice the clearance of a stubborn occupational dermatitis following the correction of a mild alimentary toxemia or the removal of a focus of infection elsewhere at the same time as local therapy is continued.

Cutaneous therapeutics cannot be divorced from general medicine. Every weapon in our medical armamentarium should be employed in curing the patient as well as the skin.

In some cases, the eruption may become so widespread that hospitalization is required. At first glance this may appear to be a costly measure, but it is often actually a saving device. In the first place, it removes the victim from the source or contact agent which caused the dermatitis, very often a difficult pathogen to discover. Secondly, he is placed under the observation of a skilled nurse who can observe the patient and prevent untoward developments from scratching, rubbing or neurotic excoriations by suitable restraining devices. The utilization of local remedies is carried out properly and sufficiently by the nurse. Any additional general measures including proper dietary regime, adequate state of hydration, intravenous therapy and laboratory investigative procedures, suitable rest and sedation may be introduced and properly applied. In these extreme cases, the total disability and severity of the illness are often mitigated by the preceding measures. Of course, most cases do not require hospitalization, but in all cases attention to general and non-specific measures are often expedient in relieving an otherwise troublesome condition.

In many of the milder cases, the workers may be allowed to continue at the plant with the addition of some protective ointments, to be discussed shortly, as well as additional cutaneous covering for protection. Within three or four weeks this may lead to the development of an immunity through the agency of a desensitization process thus preventing a neurosis in the patient from worry over his altered economic status. It should be remembered that this immunity will persist only when the worker is continuously at work. Unfortunately, if the employee leaves his job for several weeks, the immunity will wear off and an outbreak may occur after the resumption of employment.

When the patient does not develop an immunity to the materials employed in the course of his occupation after a fair trial, he should be transferred to some other section of the factory away from the irritating or noxious materials. Attempts have been made in many plants throughout the country to desensitize these individuals to the irritants to which they are exposed.

In most cases, this attempt to desensitize persons who do not, after a prescribed interval, develop an immunity, has proved both unsuccessful and impractical.

Local Therapy

WE FINALLY come to the most important phase of the treatment of contact dermatitis: the employment of topical remedies. This discussion will follow pathological development inasmuch as the only rational application of pharmaceuticals is to cope with the stages of inflammation of the skin. In the acutely inflamed lesion with erythema, slight elevation of the epidermis, possible vesiculation or bullous formation, the remedy of choice is a wet application. The comforting and soothing qualities of this treatment cannot be overemphasized. It is at the same time antipruritic, cooling and sedative, while allowing proper drainage and cleanliness. There are many useful solutions, but probably the most effective is Burow's solution of aluminum acetate diluted with 1-20 to 1-10 parts water. Others include boric acid, Thiersch's solution, Goulard's solution, and Schlotz' solution.

In the employment of wet dressings, it should be noted that the dressings are to be kept continuously moist and not, as so often happens, allowed to dry. These dressings should be continuously changed and kept wet or they defeat their own purpose. In certain types of cases, powders, lotions or pastes may be employed. As a rule, these agents are of greater value in the dry and desquamative stage. The powders act by virtue of their physical properties. The fine, compactly arranged granules cover and protect the underlying tissue from external irritation and afford an increased surface for heat radiation from the skin. This increased radiation with the attendant capillary attraction tend to remove the fluids quickly, cool the skin, contract the blood vessels and thereby relieve congestion. The powder protects, cools and drys the skin. Some of the most active powders are the minerals: zinc oxide, zinc stearate, magnesium carbonate, etc. Bismuth subnitrate, terra silica and kaolin are heavier and absorb large amounts of fluid, making them particularly valuable in the presence of a continuous fluid discharge. However, the gradual admixture of the powder with the fluid causes a caking which neces-

sitates their frequent removal and reapplication.

The vegetable powders such as starch, corn, rice and lycopodium are also valuable but should be applied with caution. The granules tend to swell, ferment, and decompose when mixed with sweat or other fluids so that they eventually irritate the skin. They should not be allowed to remain on the skin for too long a time especially in a caked condition, as they may eventually roughen the epidermis, cause an allergic eruption, obstruct the pilo-sebaceous follicles and predispose the skin to pyogenic infection. For purposes of antisepsis, boric acid, salicylic acid and calomel may be added, while benzocain, phenol and menthol are used for their antipruritic and cooling effects. Combinations of the preceding with bland ointments such as boric acid or zinc oxide, Lassar's paste, Burow's paste are indicated for the desquamative phases.

If the reactions are more exudative, lotions which are essentially fluids containing powder are employed. It is essential in these cases to apply the lotions with the proper technique. They should be shaken and spread on the skin with cotton or a soft camel's hair brush, and allowed to dry. The evaporation of the liquid cools the skin while the coating of powder continues its action. Glycerine is frequently added for the benefits of its hygroscopic action and also to keep the powder more continuously in contact with the skin. The proportion of glycerine is reduced or omitted entirely in cold weather because of its tendency to make the skin cold and clammy. However, more glycerine is added in warm weather, but its strength is rarely above ten per cent, when used for acute inflammations, as it may become irritating. The fluids employed are usually lime water or rose water. When these prove to be too drying, distilled water is substituted.

Alcohol is sometimes added for antiseptic, cooling and drying purposes.

No field of medicine requires the services of a scientific pharmacist more than does dermatology. The quality of the ingredients as well as the care with which the prescriptions are compounded are of great importance. A simple, and probably the most frequently used and useful lotion is the calamine and zinc oxide lotion. Even this simple lotion may show a difference in appearance and in its effect on the skin when not properly prepared.

In this preparation, chemically pure zinc oxide and a fine pink prepared zinc carbonate (prepared calamine) should be sifted through a fine sieve.

The mixture should then be rubbed with glycerin and a small amount of the official limewater added to make a fine paste. Then the rest of the limewater and rosewater are added. The zinc oxide and calamine act as a powder after the waters have evaporated and the glycerin soothes and keeps the powder in contact with the skin. When menthol and phenol are prescribed they should be rubbed together and a small amount of alcohol added to aid the liquefaction, and for antipruritic and cooling effect.

The rupture or tearing open of the vesicles results in the escape of fluid on the surface of the skin.

When this desiccates, crusts result. In crusted cutaneous conditions it becomes necessary to remove the crusts before attacking the underlying pathological tissue. Watery solutions applied as wet dressings are not only efficient cleansing agents but they also act to reduce inflammation. A pad containing several layers of gauze is soaked with a weak solution such as a one to ten Burow's solution, a 2% boric acid solution, or a saturated solution of magnesium sulphate, and then applied to the inflamed crusted surface. The liquid softens and macerates the crust which gradually become detached. The gauze should originally be saturated and more fluid frequently added to maintain sufficient moisture. In acute conditions the gauze should not be covered with an impervious material like gutta percha or rubber tissue, as this prevents evaporation and causes retention of heat which results in irritation of the already acutely inflamed skin. On the other hand, it may be desirable to stimulate rapid softening and breaking down as in the presence of chronic inflammations with firmly adherent thick crusts, or when hasty necrosis is desirable as for furuncles. In such cases, the impervious covering may be justified. Subsequent to the removal of the crusts the lesions should be treated as suggested for acute vesicular eczema.

Cutaneous conditions of several week's duration usually show cellular infiltration, changes in the collagen, hypertrophy of the rete pegs, acanthosis, hypertrophy of the epidermal cells and especially of the cells of the stratum corneum resulting in parakeratosis and hyperkeratosis. The skin appears dark red, thickened, and scaly. Both lotions and pastes act well in such cases. The latter are fat preparations containing high percentages of powder.

When spread on the skin, the fat covers, protects, softens and lubricates the skin. The incorporated powders favor absorption of secretion, radiation of heat, and relief of congestion. Lassar's paste contains approximately 50% of powder, equal parts of zinc oxide and powdered starch, and 50% of fat, usually vaseline. This makes a thick paste that is more or less difficult to spread and it is often more practical to reduce the powder to 25 to 40% without affecting the efficiency of the preparation. Salicylic acid, in 1% to 3% strength is frequently added as a mild antiseptic, to prevent fermentation of the discharges and to dissolve the abnormal surface scales. When secondary pyogenic infections have caused pustule formation, an antiseptic like ammoniate of mercury may be added to the above preparations.

Cold cream makes a splendid fat base for pastes and enhances the value of the paste by cooling and reducing congestion.

Creams are fatty preparations that contain water-usually about 20%. The fat covers the skin, the water in the cream evaporates, the cream then absorbs water from the skin, especially if there is inflammation, reducing the temperature of the skin. This action is the reason they are called cold

creams. Many of them contain anhydrous lanolin or aquaphor to aid in taking up water from the skin.

Petrolatum makes a good base as it does not decompose, while wax may be added to make creams firmer. Since they tend to become rancid, it is necessary to add a mild antiseptic such as boric acid to the fat and water, while some such perfume as rose water may be added, as desired, for aesthetic purposes.

The chief uses of creams are to cleanse, to protect from external irritants, to supply oil and fat where the natural secretions are deficient, and to serve as a base for other applications. They are spread on the skin and covered with a powder or gently rubbed into the skin, making it soft, smooth and lustrous. They are excellent non-drying solvents and may be used for dry, scaly, chapped and cracked skin.

For chronic lesions of the skin, ointments are most frequently employed. These consist almost entirely of fat with drugs added for medicinal action. When fat is spread on the skin it covers it like an impervious material and prevents escape of fluid and causes retention of heat. The ultimate result is irritation which stimulates the sluggish skin. Circulation is improved, absorption is favored, resulting in a tendency towards the formation of new, normal cells. Surface scales and crusts are also softened and rendered more easily removable since the surface cells swell as a result of the penetrating action of the fat.

Drugs employed in ointments for specific medicinal purposes are varied. Tar, sulphur and resorcin are commonly employed for anti-pruritic, antiseptic and keratolytic action; salicylic acid to dissolve hyperplastic tissue; chrysarobin and pyrogallic acid as reducing agents and caustics. Many of the drugs incorporated in the ointments act as keratoplastics when present in small amounts, and as keratolytic agents when more concentrated.

As ointments are used in chronic conditions and where stimulation is desirable, rubbing in of the preparation is advisable. In old, obstinate, resistant, thickened patches, the action of an ointment may be increased by covering it with an impervious material such as oiled silk. If the irritation is excessive an acute inflammation may supervene.

This result is often desirable and is subsequently treated as an acute eczema.

When surface action for a dry condition is the aim, petrolatum is useful. Lanolin or goose grease may be added to increase penetration, anhydrous lanolin is used for moist areas, and wax is incorporated when the preparation needs stiffening.

Little has been said concerning the treatment of various parts of the body. However, therapy should be modified depending on the thickness, hydration, pilosebaceous apparatus, adjacent organs and blood supply of the region involved. Naturally, the treatment of a dermatitis of the hand presents different problems in management from one confined to the face.

In the local treatment of contact dermatitis, other modalities are often of use. Some conditions are beneficially affected by applications of ultraviolet rays. In more stubborn cases, the usage of x-ray therapy may help initiate a remission. Much benefit may accrue from the careful application of properly measured doses by the experienced dermatologist who correctly diagnoses the clinical lesions and is able to interpret these in terms of their histopathology. This knowledge is then utilized in order to obtain the best biological effects without injuring the integument. In conclusion, it should be stated that success in the management of a case depends upon the proper selection and application of the products employed always keeping in mind the underlying pathology of the lesion.

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Silicosis in Switzerland

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UNTIL 1931 little had been heard of silicosis in Switzerland although Wegmann published a detailed study of the question of dust in factories in 1894 and, in 1906, Zangger drew attention to cases of pneumoconiosis among sandstone grinders, who were at first treated for tuberculosis, and in 1912 placed two patients in a sanatorium.* Staub examined these two cases in 1916, clinically and radiologically, and considered they were classical cases of silicosis. These were probably the first two cases of silicosis to be compensated in Europe. About this time Zangger recommended the replacement of sandstone by emery wheels along with adequate dust removal by exhaust ventilation. Interest then died away and silicosis was not even thought of in the preparation of concurrent compensation insurance.

Suddenly, in 1931, numerous cases of silicosis occurred among sandblasters in steel works and in the manufacture of scouring powders. At this time the pathological institutions had no positive experience with silicosis so that the disease was not then placed on benefit compensation by the Swiss government. But, on January 1, 1932, the insurance law was amended authorizing benefits, at official discretion, for pneumoconiosis, meaning specifically diseases caused by dust and not those which might be aggravated by it. This act provided for insured persons who were, at that date, undergoing treatment for pneumoconiosis and suffering from total invalidity or totally and permanently incapacitated for work. The law was made retroactive to include those employed as of January 1, 1931. Thus the same benefits became available for silicosis as for other occupational diseases, and granted in full for both unemployment and medical costs, the only reservation being that the insured did not possess the right of instituting legal proceedings against decisions regarding him by the Fund.

In the course of the last few years, the government has declared silicosis to be an occupational disease subject to compulsory insurance by adding "silica or quartz (silicosis)" to the schedule, effective May 1, 1938. The insurance is not limited to certain trades, as in Germany.

About 200 cases of silicosis have been notified (reported) since the National Fund began to pay compensation for this disease (1931). They occurred under the following exposures: sandblasting, cleaning of castings, grinding on sandstone wheels, tunnel and mining construction, quarrying and cutting of stone, the pottery industry, and the manufacture of scouring powders.

Not a single case has been found in which a pneumoconic complaint was not due to silicon dioxide. The observations of others in regard to *silicates* and in particular *sericite* have not been confirmed in Switzerland, although workers exposed to sericite, free from quartz, have not been examined. However, the observations of others have been confirmed in regard to the significance of the proportion of quartz in the dust, size and distribution of particles, duration of exposure, etc. Depending upon conditions, the disease may appear within a few years or as long as 30 years or more of exposure. The shortest exposure recorded, which led to death, occurred in a sandblaster, the duration but one year. Autopsy confirmed the diagnosis.

Special attention was given to the action of sericite upon *slateworkers* where quartz was also present from 5 to 20%, and carbonated lime to 20%. But silicosis was found not to occur among slateworkers under 20 to 30 years' exposure and then the condition is accounted for by the small proportion of silicon dioxide contained.

Cementworkers' lungs sometimes show on their surface a "honey-combed" network, probably of slight sclerosis. No cases of serious anthracotic changes, siderosis, or aluminosis, or from the effects of emery wheels (which contain only 1 or 2% of silicon dioxide) have been encountered.

In the light of experience had, the policy has been adopted of studying the occupational case history very carefully—"a policy which has given good results," especially in controversial cases, with and without tuberculosis, and where the radiographs have shown marked increase of the pulmonary network, with mottling and a pronounced hilus, with the function of the heart and lungs very much reduced. These workers were emery wheelmen and smelterers who had worked for many years, with exposure only to very small quantities of quartz particles. Some cases were shown by postmortem to be pure tuberculosis; one was bronchial carcinoma; and one lymphangioendothelioma of the hilus glands. In the former case, sidero-anthracosis and, in the latter, serious anthracosis were found.

On the question of the relation between *silicosis and tuberculosis,* the Swiss experience (of 200 cases), particularly that of Uehlinger in his anatomo-pathological studies, lead to the conclusion that second degree, and especially third degree, silicosis may favor the outbreak of tuberculosis. Silicosis may also hasten the development of a pre-existing tuberculous condition, or revive and stimulate pre-existing foci, particularly post-primary tuberculous foci. In this respect, pneumoconiosis due to mixed dust is probably more dangerous than pure silicosis. In one case, Uehlinger proved irrefutably that silicosis due to mixed dust, though so slight it could not be diagnosed clinically, had revived former post-primary foci by means of very active dust granulomas. The development of superimposed tuberculosis appears to vary according to the worker's occupation.

Nearly half of the Swiss autopsies have revealed, however, the entire absence of tuberculosis, even in cases of serious silicosis, thus contradicting the view of various French doctors.

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* From report presented at the 2nd International Conference on Silicosis, Lucerne, September 3, 1938; Abstracted by EMERY R. HAYHURST, M.D., from *International Labour Review,* 31,1:46-53 (January) 1939.

The Swiss findings regarding radiological and clinical findings of silicosis and silico-tuberculosis do not differ from those of numerous foreign writers. On the other hand, radiographic examinations of silicosis by occupations emphasizes special findings which are not connected with the several stages of the disease. Thus, in sandblasters, the mottling of the lungs ("snow-storm" design) is finer than in any other group, and rarely contains nodules even in fatal cases. Large nodules are more common among miners. Stonecutters and slateworkers show fairly large mottling and clearly outlined "lead shot" lungs. Pneumoconiosis due to mixed dusts produces more blurred shadows. Pneumothorax was encountered on several occasions, and, in three cases, traction-diverticulum of the esophagus was revealed. Thus, to sum up, error can be effectively guarded against by a critical occupational history, conditions of work, history of the disease, and all clinical and radiographical findings.

In about one-third of the cases, the disease did not appear until after cessation of the work which caused it, the interval varying between a few months and several years.

Regarding the *therapy of silicosis,* rest at a certain altitude has resulted in effective arrest in cases of slight or medium gravity, with and without tuberculosis, and has made possible the worker's return to partial or full employment. Bronchitis and circulatory complaints may be treated in the ordinary way.

The aspect of the *evolution* of the disease which has impressed the Swiss most forcibly is the amount of variation found between different trades. "Quite a number of cases in which the disease was detected at the outset have remained stationary for long periods, and usually the patients have recovered their working capacity in full or in part."

As to the form of *compensation:* Where possible, every patient is placed for a few days in a hospital in Lucerne for examination. In nearly one-third of the cases notified, the diagnosis of silicosis had to be abandoned. According to Teleky only one-fifth of the cases notified in Germany from 1929 to 1932 were admitted to insurance benefit. When a case of silicosis is recognized as one for insurance, the National Fund must grant the victim all the statutory benefits. "There are not clauses limiting the grant of benefits to serious cases, or imposing other restrictions." But, diseases such as compensated cardiac deficiency or serious pulmonary tuberculosis are not granted benefit where only slight silicosis is found. However, cases of this kind are very rare.

Tuberculosis following upon silicosis, on the other hand, entitles a patient to full benefit. When pre-existing tuberculosis is aggravated by silicosis, the Fund is authorized to reduce the cash benefit but not the expenditure on curative treatment. This question of cash benefit sometimes presents serious difficulties and the decision is particularly difficult in the case of aged persons where incapacity may be due to several possible features other than silicosis — then it is usually a matter of personal opinion.

As long as the patient requires treatment, the National Fund pays the cost of the cure and sickness benefit. Where permanent invalidity seems probable, the person receives an invalidity pension which varies from 25% of wages to full wages. The same is extended to surviving dependents.

The National Fund has adopted certain measures *to combat silicosis:* Inspectors make regular visits to all undertaking where workers are exposed to the risk of contracting the disease; they are instructed to have all workers examined before employment; exposed workers are subjected to periodic medical examinations; the doctors are given a standard form to be adhered to; government officials carefully examine the reports and radiographs and decide, for doubtful cases, the type of work to which the subject may be put in the future and the date of his next examination; a pamphlet setting forth the problem of silicosis, in simple, clear terms, has been widely distributed; and the cost of preventive medical examinations is levied on the managements. By Section 60 of the insurance law: "The employer or his representative shall adopt, with a view to preventing sickness and accidents, all measures which experience has shown to be necessary and which circumstances and the progress of science make practical." The National Fund may order the adoption of any useful measure, but the parties may appeal to the Federal Council within 20 days.

One measure adopted is to transfer workers having dangerous exposure, after a certain period, to a department where there is less or no risk. "The systematic application of medical and technical prophylactic measures is combination has resulted in a decline of the disease. The problem of effective protection for miners and stonecutters, however, remains to be solved."

Arthritis and Injury

—*and their Relation to Injury and Arthritis*—

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ARTHRITIS can hardly be called an industrial disease, but it is certainly almost a universal disease and it has surely a great effect upon the production in industry, which includes our own industry, so that we are especially interested in it.*

A long time ago, when I started out in the stockyards, I noticed a lot of fellows complaining of pain in the joints and principally of pain in the back and when I had seen enough I began to ask questions about in what type of individual, what aged individual this condition occurred. For the most part it occurred as the result of slight trauma

* Read before the Congress of Railway Surgeons, September 19, 1938, at the Palmer House, Chicago.

and then persisted. It persisted beyond all relation to the trauma which caused it, and when I came to look over the record of the patients, and I had a much better memory for that in those days than I have now, I was very much impressed with the fact that a large percentage of these patients who had increase in joint pains and in back pain were over 40; that when a young individual came in with an injury, the injury was sufficient to account for the symptoms, but in a large percentage of cases in the older individuals the injury was insufficient to cause the severity and persistence of symptoms. This should mean something.

We have described three types of arthritis: the atrophic, the hypertrophic and the traumatic, about which there is very little said. The atrophic type I think we are all familiar with as being caused by infection; that has been pretty well established. It more frequently happens in the young than in the elderly patient. There is almost always a focus of infection that can be found with care and persistence. This infection is the cause of a constitutional disease with joint manifestations and I think in this discussion we can practically eliminate rheumatoid arthritis, because these cases usually are polyarthritis. But atrophic arthritis is a constitutional disease with joint manifestations, and when we began to treat it as a constitutional disease with joint manifestations and treated it as we do tuberculosis and other infections by putting the patient to bed, free from other trauma and irritating causes, we got somewhere with it.

Hypertrophic arthritis is a condition which occurs in patients who are past or at middle age. It may be the result of toxemia, and when I say toxemia I mean any form of toxemia. Infection certainly ought not to be overlooked, but I think in many of these cases the toxemia is due to some metabolic disturbance of which the patient has not been conscious for over a period of years. For instance, gout. Most of us learned the diagnosis of gout from Little Lord Fauntleroy as due to wine drinking and eating beef, in an old man, but this is not true. It is just as truly a metabolic disease as diabetes. I have seen it in a boy of 14 with a blood pressure of 120 to 170 in the course of 24 hours. I have seen it in a long distance swimmer of 18 who was a fine physical specimen. I have seen it in a young man in active football training who was six feet two inches tall, and in the middle of football season he had gout. The uric acid in gout is not always highest at the time the attack occurs, so many tests may be necessary. However, this is only one of the toxemias which may occur.

When I talk about toxemias before an audience of internists, they ask me, what are these toxemias; what is allergy; what is an allergic response or reaction; why can one man eat strawberries and get no reaction and another man break out with hives?

Many of us have idiosyncrasies or factors of safety which are higher in the adolescent than in older life when the fires do not burn as brightly, when there are hidden infections, when there are worries and overwork and fatigue. All of these things cause toxemia. Over a period of years this amount of toxemia in excess of normal seems to have a tendency to settle out in what? In the thing that no toxins affect, producing pain in the ligament. The ligaments are the first things to give a cry for help when toxemia and infection exist.

Why do we have these severe backaches and pains without joint symptoms in many of our acute infectious diseases? Why, when we sit down in a draft, do our ligaments stiffen up and this did not happen to us 20 years ago? Those are questions to ask ourselves. It is certain in my mind that we have many forms of toxemia about which we know nothing at present.

Our textbooks teach that gout destroys, by deposit and extension, the bone surrounding the joint. One of our patients, a man, had a slight injury to his knee; the knee was just bumped on the side. He had rather sudden pain which did not lay him up, but it continued to get worse the next day and then became acutely swollen but was not red or tender, although the joint was somewhat tense. On x-ray there was found a gouged-out place in the knee. His uric acid was 8.2 mg. per 100 cc. of blood. He had been laid up for a good many weeks and when he was put on a diet with atophen, he promptly cleared up, went back to work in two weeks. He worked six months and then had pain again. That pain was in a different place from that he had before. He finally quit work and came back for another examination. At that time he had a small destructive process on the edge of the joint, and this patient subsequently developed a very acute tuberculosis in this joint and finally had the leg amputated.

How much damage had the gout done to the substance of the joint? I believe this joint was not infected at the first injury.

ANOTHER patient was a pure case of traumatic arthritis who had had one joint disabled to the extent that he had been retired, on a pension. He had had a fracture previously but he weighed 225 pounds and had been working as a fireman for years but had been retired because of the condition of his knee. There is one thing we can be sure of in these cases. When we see evidence of arthritis in the x-ray, it is ancient evidence. The arthritis that we see in the x-ray is just like the osteomyelitis we see on the x-ray. The damage has been done. When we see it on x-ray we are sure it has been there a long time and we know that the arthritis is not a result of the recent injury, but look out. That arthritic joint with a slight injury will give tremendous disability, whereas if there had been no arthritis there might have been only a few days of disability. An operation was performed upon this man's knee and many pieces of debris removed. The cartilage was not atrophied. The rough surface was completely cleaned off so that there was nothing but a smooth cartilage underneath.

As you may know, fat women's knees have a great tendency to develop arthritis. Why? Because those are the joints that are subject to tremendous strain, and frequently a good deal of cross strain because these women walk with their knees bent, and the knee joint, which bears the cross strain, will develop arthritis. We see the same thing in these old arthritic spines.

These arthritic spines cannot be tampered with. Such a back must be protected at the time we see it, not months afterward. It should have no physiotherapy for months. What that patient needs is not to be jiggled over a road in a street car or automobile. It should be in bed where it gets rest, because slight trauma on this type of joint will cause an inflammatory process to exist month after month and nothing you can do will stop it as long as that patient keeps on traumatizing it. With an arm you can put it in a cast, but you cannot immobilize the spine until you put the patient in a horizontal position.

I have been interested in some experiments concerning the reaction of cartilage to trauma. We followed a number of traumatized and infected joints and put foreign bodies in. We found that when cartilage was interrupted at the weight-bearing surfaces, the pieces of cartilage degenerated. When the cartilage is removed or broken, there is no effort towards bridging that gap as there is in an epithelial surface. If we strip a piece of cartilage up from the matrix, it will not reheal; it will hold on to its pedicle but if taken off completely will not regenerate. If part of the matrix is left on it will regenerate to some extent. In a case where the cartilage and bone both were cut, there was a heaping up of the hyaline cartilage as a result of the matrix throwing out new cells. In this way if there is a depressed fracture, the cartilage leaves off on the surface, but if it is gouged out it will never remain smooth. As long as there is no rough regeneration of cartilage the joint will slide normally.

We tried to produce arthritis in dogs. We were entirely unable to do so until the internal lateral and crucial ligaments in the knee were severed and not resutured, but the skin closed and the dog allowed to run and walk and play. This dog could go fast but he was very careful when he turned the corner. He always turned so that the weight of the turn was thrown on the intact ligament.

We have operated upon something like 60 of these arthritic joints and I have never seen a true hyper- or atrophic arthritis. I have never opened a joint that did not show some of the symptoms described for both of these conditions. What can trauma do to these cases? I myself have had a fracture of a scaphoid of the wrist. The result is pretty good and the function is good, but I am not a carpenter. I do not have to use this wrist for constant heavy lifting motions that cause slight jarring trauma many times in the course of a month. But I have had a patient who was a carpenter and had the same type of fracture, and he is now having a breakdown with disability. You can hear grating around the wrist. It is a real traumatic arthritis, with absorption of bone. When the new bone was removed, the patient got a comfortable wrist with which he was able to return to work because the irritating cause, which was a rough cartilaginous surface, had been removed. This rough cartilaginous surface constantly irritated and formed a true traumatic arthritis. In contrast to this case, we had, some 12 years ago, a man with a broken posterior condyle of the knee. That man is still working as a switchman; he jumps on and off cars without any difficulty. The reason he can do that is because he happened to be a young man when this fragment was replaced; circulation was reestablished, the cartilaginous line was continuous, and it filled in without roughening. The patient has no disability in the joint because there is no chronic irritation.

Now, I feel that many of us for a long time have felt that arthritis and trauma had no bearing upon each other; and I feel that we ought as doctors to be very careful about the patient who receives an injury to the joint, whether it be in the back, the knee, the hip, the shoulder, that causes a moderate amount of swelling and pain which the patient says is not so severe that he cannot get around but continues and continues. If there is evidence of arthritis in the x-ray, then that joint should be immobilized, not necessarily with a cast. I like traction much better than a cast. Casts do immobilize if properly put on, but they do not remain set, and weight-bearing members can only be rested by putting the patient in a horizontal position, by putting on traction that relieves the joint of part of its irritation. It is better to completely immobilize, because these joints get stiff very rapidly. Rest in bed is important, and rest in bed first, not six or eight or 10 months after the patient has been coming for treatments, which will never do him much good as long as he traumatizes the joint getting to and from treatment.

Discussion:

DR. WILLIAM HOLMES, Chicago: The subject of arthritis offers many opportunities for differences of opinion because it can be approached from so many different angles.

Dr. Magnuson approaches it from the standpoint of the bone surgeon, an approach which naturally emphasizes certain features such as the dynamics of motion and weight bearing which are poorly understood by men like myself who approach it from the standpoint of internists, pathologists, bacteriologists or chemists. Despite the impressions gained by views of the subject from different angles, there are certain fundamental facts upon which I think we can all agree.

We can agree, for instance, that injury to a joint may be the most important determining factor in the localization of pyogenic bacteria, such as the gonococcus and the pneumococcus. It is significant that this form of acute suppurative arthritis is mono-articular, involves by preference the knee or ankle and is fortunately relatively infrequent. It is infrequent not because gonococci and pneumococci do not get into the blood stream frequently, because unquestionably they do, but because they do not survive there or in the tissues unless conditions are suitable for their growth. The normal joint, like the normal heart valve, is resistant to their implantation. If this were not true, gonorrheal arthritis would be polyarticular and would not attack by preference the joints frequently subjected to trauma.

However, when we use the term "arthritis," it is not the acute suppurative form which first comes to mind. Rather it is the chronic form conventionally divided into the atrophic and hypertrophic types, the latter of which is really an arthropathy. With reference to these, I think surgeons and internists can agree that here we are not dealing solely with a disease of joints but with a constitutional disease, the result of chronic infection. That it is a constitutional disease is indicated by the fact that the atrophic form is generally found in persons of a certain physical type: asthenic, flat chested, visceroptotic, hypotensive, immature adolescents, preponderantly girls. Its constitutional nature is further indicated by the coexistence of anemia, a lowered rate of metabolism, a low surface temperature, constriction of the capillaries, sweating of the hands and feet, chronic constipation, and an almost universal distribution of joint symptoms. Although both large and small joints are involved, the most serious disability is usually due to involvement of the knees and spine; in other words, the weight bearing joints. This to me indicates operation of three forces:

(a) constitutional physical inferiority;
(b) chronic infection;
(c) physical strain or trauma.

In the hypertrophic type of chronic arthritis, the constitutional make-up is quite the opposite of the atrophic. Here we are dealing with well nourished, usually obese adults in the fifth or sixth decade in whom evidence of the infectious nature of the process is not so clear cut. Curiously, however, here we again find that it is the knees, the spine and the fingers that are most seriously affected, indicating that however the two types may differ in pathogenesis, there is at least one factor common to both: a factor which appears to be physical strain or trauma.

The importance of circulatory changes in the genesis of arthritis is indicated by the occurrence of bone changes in persons suffering from pulmonary and cardiac disease as a cause.

In the atrophic type, circulation is impaired by capillary constriction; in the hypertrophic type by the vascular changes of advancing age.

The importance of trauma in the genesis of hypertrophic arthritis is exemplified by the changes observed in tabes. Here due to loss of deep sensibility, there is hyperextension of the knee joint, relaxation of the capsule, faulty weight bearing and as a result continuous trauma to the joint leading to bony overgrowth and finally to complete disorganization of the joint. The tabetic actually pounds the joint to pieces.

In the control of arthritis, therefore, attention must be directed toward three major factors:

(1) constitutional pre-disposition;
(2) infection;
(3) trauma.

A broad viewpoint rather than a slavish adherence to a ritual requiring administration of sulphur, gold salts or vaccines is, therefore, important.

Of the three major factors concerned, medical men like myself are most prone to ignore or neglect the factor of trauma.

QUESTION: I was interested in this paper and discussion because I have a case of traumatic arthritis in a woman weighing 270 pounds, and about 75 years old. I should like to ask whether local applications, such as heat or cold, would be advisable.

DR. PAUL B. MAGNUSON, Chicago (closing): Yes, I think heat probably is one of the most valuable assistants we have in the treatment of traumatic arthritis. Any kind of heat is good; I do not care whether it has a wire attached to it or not. A good hot towel frequently applied does well. I think inductotherm for the deep treatment of joints is advisable if we are sure there is no infectious process in the joint, but I think inductotherm or diathermy do distinct harm in an infected joint or a joint condition which is the result of any kind of acute or subacute infection.

The Use of Sulphur Dioxide

—*As a Refrigerant*—

WM. D. MCNALLY, A.B., M.D.,
Chicago

THOUSANDS of workmen are exposed yearly to the irritating effects of sulphur dioxide during the manufacture of sulphuric acid, refining of petroleum, fumes from metallurgical works, in its use as a bleaching agent, in the manufacture of cellulose, and in the filling, installation and repair of refrigeration units in homes and in commerce.

Since there are many more workmen exposed to sulphur dioxide in the refrigerating industry than in other uses of this gas, I shall stress in this paper this phase of the use of sulphur dioxide.

One of the great and pressing problems that has confronted man from early times has been the conservation and preservation of food supplies. In time of plenty, he attempted to preserve food to have available in time of need. An early method used, was that of air drying by means of the heat of the sun. Later, a more rapid method was developed by the use of fire and smoke. At a still later period, salt, spices, ice, chemical preservatives and canning of food were used. In the colder regions, snow from the mountains and ice from the rivers and lakes were utilized. Ice was cut in blocks and hauled to ice-houses where it was prevented from thawing by the use of sawdust. But the purity of this ice was questioned by the health authorities and in consequence the artificial ice industry developed. In rural communities, running water from streams is still used for preserving milk, butter and cream.

In the last 30 years the canning industry has made great progress in the preservation of the many varieties of foods. The cold storage industry during this time also has been able to preserve all manner of foods and meats, so that the American people have available, fresh foods in all seasons. The latter industry made use of mechanical refrigerants using ammonia or carbon dioxide to reduce the temperature.

In the first efforts to produce mechanical refrigeration, a light grade of gasoline was used. It was discontinued, however, because of the fire hazard. Later, ammonia, carbon dioxide, sulphur dioxide, methyl bromide, methyl chloride, ethyl bromide, and ethyl chloride came into use. Because of the dangerous properties of methyl chloride, which caused many cases of illness and a number of deaths, sulphur dioxide came into extensive use since 1929. Being an irritant gas, if a leak develops in the refrigerating system, this gas becomes its own warning agent.

MacIntire, in his book entitled, "Refrigeration Engineering," makes the statement: "In 1876, Raoul Pictet, devised the sulphur dioxide compressor, using water to cool the pistons and piston rods as well as the cylinder." This seemed to be the generally accepted beginning of present day sulphur dioxide compression refrigerating ma-

chine. In this country, from 1900 on, there was considerable experimental work on a small sulphur dioxide machine that could be used in the home, or for other fractional tonnage work. During this period, the "Audiffren" machine was devised by Abbe Audiffren of Grasse, France.[1]

SULPHUR dioxide has been used as a refrigerant by many manufacturers because it was found to be non-explosive and non-inflammable. Due to its being its own warning agent when a leak developed, it became the ideal refrigerant. Owing to the fact that it finds considerable uses other than refrigeration, sulphur dioxide is a low-cost chemical. When dry, it will not react with any of the metals usually found in machine construction. The great disadvantage of sulphur dioxide, however, lies in its corrosive nature especially when it becomes mixed with water.

Sulphur dioxide is found in volcanic gases and in spring waters of volcanic regions. It can also be detected in the air of manufacturing communities where large quantities of coal are used for fuel. It is found in the smoke of foundries in such small quantities as to be of no consequence. In railroad terminals and tunnels it is sometimes in concentrations sufficient to be irritating to the throat. Air containing 0.07% of sulphur dioxide is unbreathable, the gas not having a toxic effect but causing a spasmodic contraction of the air cells of the lungs. The air of the city of Chicago* contains from 0.000031 to 0.000136% of sulphur dioxide; the former amount was found at State and Madison streets. Berlin air contains 0.000035% of sulphur dioxide; London, 0.00039%; Glasgow, 0.00042%; and the vicinity of Salt Lake City, Utah, from 0.001% to none. It may be detected by the sense of smell if present in ratio of more than one in 150,000 parts of air. Schade[2] reported that sulphur dioxide present in the air in the city of Pittsburgh is too small to cause concern from a hygienic standpoint. Meiter and Traubert[3] report that the average amount of sulphur dioxide content of the air at the Pittsburgh Experimental Station of the Bureau of Mines was 0.16 parts per million. The maximum, 1.1 parts per million, was found on two days; less than the minimum detectable (0.1 part per million) on 141 days. They found that the concentration tended to be higher on foggy or smoky days.

Sulphur dioxide may be manufactured from burning sulphur, or roasting various sulfides. Moliter[4] advocates heating to 800°C a mixture of $CaSO_4$, coke, powdered shale and clay, to obtain sulphur dioxide.

Aqueous solutions of sulphur dioxide slowly decompose at room temperature forming ultimately H_2SO_4 and S. Sulfurous acid forms three classes of salts: normal, as sodium sulfite, Na_2SO_3; acid, as sodium bisulfite, $NaHSO_3$; and meta, as sodium metabisulfite, $Na_2S_2O_5$. It reacts with Zn, Fe, Sn and Cu to form hyposulfurous acid, $H_2S_2O_4$. Zn in the presence of HCl reduces H_2SO_3 to H_2S.

* A Survey of Sulphur Dioxide Pollution in Chicago and Vicinity, *University of Illinois Bulletin*, Vol. 36, No. 37, January, 1939.

Sulphurous acid and sulfites are active reducing agents because of their capacity for oxidation to sulphuric acid and sulphates. Sulphurous acid reacts as a reducing agent toward other acids, except hypophosphorous, phosphorous and hydrosulfic. Hydrogen peroxide oxidizes sulfites to sulfates. The free halogens, Cl_2, Br_2, and I_2, react with SO_2 to form the corresponding halides. The halogen oxyacids, with the exception of $HClO_4$, are also reduced to the halide.

SULPHUR DIOXIDE
Physical Properties

Chemical formula	SO_2
Molecular weight	64.06
Color (gas and liquid)	Colorless
Odor	Characteristic, pungent
Melting point	98.86°F. (—72.7°C.)
Boiling point	14°F. (—10.0°C.)

Temperature °F.	Density (Water=62.4) Lb/cu. ft.	Specific Gravity (water=1)
–40	95.79	1.535
0	92.42	1.481
40	88.81	1.423
80	85.03	1.363
120	80.90	1.296

Density of Gas (0°C. and 760 mm.)

Referred to Air	2.2638
Gms. per liter	2.9269
Lbs. per cu. ft.	0.1827

Critical temperature	214.82°F. (157.50C. 0.05°)
Critical pressure	1141.5 lbs. per sq. in. abs.
Suction pressure at 5°F.	11.81 lbs. per sq. in. abs.
Head pressure at 86°F.	66.45 lbs. per sq. in. abs.
Volume of saturated vapor at 14°F.	5.164 cu. ft. per lb.
Volume of liquid at 14°F.	0.01096 cu. ft. per lb.

Solubility in 100 parts of cold water—22.83 at 0°C. (32°F).
Solubility in 100 parts of warm water—4.5 at 50°C. (122°F).
Solubility in 100 parts of hot water—0.58.

Inflammability: Non-inflammable.
Exposibility: Non-explosive, will extinguish fire.

Sulphur dioxide is readily soluble in water with evolution of heat. At 20° the solubility is 10.64 g./100 g. of water. Acetic acid dissolves more than 300 volumes of SO_2 at 0° and 760 mm. The minimum solubility in H_2SO_4 is reached at a concentration of 85.8% in which 2.80 g. of SO_2 will dissolve in 100 g. of the solvent. The solubility increases with either a decrease or an increase in strength of the H_2SO_4.

Detection

A SOLUTION of sulphurous acid is detected by its odor and by its decolorizing action on $KMnO_4$ or I_2. A mixture of iodic acid, HIO_3, and starch is turned violet to blue by traces of SO_3. The color is destroyed by an excess of the SO_3. Sulfites are distinguished from sulfates by failure to precipitate with $BaCl_2$ in presence of HCl. Sulfites may be converted by Zn to hyposulfite, S_2O_4, which may be detected with methylene blue. If zinc nitroprusside is exposed to ammonia vapors, then to SO_2, a rose-red color will appear. If a 0.01% solution of Fast Blue R is added dropwise to SO_3, at first a violet, then a yellow color will appear. The test is sensitive to 1:175,000. Sulfide and OH interfere; S_2O_3 and polythionates do not.[5]

It is determined, gravimetrically, by oxidation to sulphuric acid, precipitated by barium chloride, dried and weighed as barium sulphate, or volumetrically, treated with an excess of iodine and

the excess titrated with a normal solution of sodium thiosulphate.

Sulphur dioxide may be absorbed by foods if there is a leak in a refrigerator, but most of it is removed by the cooking of the food. It has been used for many years as a food preservative. Many dried fruits contain sulphur dioxide. Molasses contains a large amount of this preservative to keep it from fermenting.

It will be noted from Table I that the least detectable amount of sulphur dioxide is from three to five parts per million. When this amount is increased from eight to 12 parts per million, there is an immediate irritation of the throat. When it reaches 20 parts per million, there is an irritation of the eyes. When inhaled, it affects the respiratory tract.

TABLE I.
PHYSIOLOGICAL RESPONSE TO VARIOUS CONCENTRATIONS OF SO_2

	Parts of SO_2 per million parts of air.
Least detectable odor[1,2]	3 to 5
Least amount causing irritation to the eyes[2]	20
Least amount causing immediate irritation to the throat[2]	8 to 12
Least amount causing coughing[2]	20
Maximum concentration allowable for prolonged exposure[2]	10
Maximum concentration allowable for short exposure, one-half to one hour[2,3]	50 to 100
Dangerous for even short exposure[3]	400 to 500

1. U. S. Dept. of Interior, Bureau of Mines, Bulletin 98, 1915.
2. FIELDNER, A. and KATZ, S.: *Eng. & Mining. Jour.*, 107, 693, 1919.
3. LEHMANN: *Arch. f. Hyg.*, 18, 180, 1892.

In the inhalation of sulphur dioxide there are two considerations, first, those results due to a single or repeated exposure to a high concentration and second, the possibility of a cumulative effect, due to continually breathing small concentrations.

Ogata[6] reports that 0.4% for four hours caused disturbance, and that with 1% all animals died in a short time. Lehmann states that doses of 0.03 to 0.04% can have great effect upon a person unaccustomed to it, but others would not notice it. Rabbits, guinea pigs and pigeons, in all 40, in an atmosphere of 0.5% for a month, six to seven hours daily, the air in the chamber being changed every half hour, at first sneezed, and seemed to be pinched, had copious flow of tears and restlessness, which disappeared at the end of the second week. During the 30 days—one rabbit died in 18 days, one guinea in five days and another in 15 days. In all three animals, the blood was of a dark red color and the lungs contained small red spots. The blood from all animals had a color darker than normal.

Sulphur dioxide even in weak concentrations, acts quickly upon the mucous membranes, particularly on those of the respiratory system. The strength which can be tolerated has been fixed by Villaret at 0.07 per thousand, but on the other hand it varies with indviduals and with the degree to which they have become inured. As previously stated, Ogata and Lehmann have shown that for individuals unaccustomed to the gas, a strength of 0.02 to 0.03 mgm. per thousand is hardly borne, and that a strength of 0.04 mg. causes embarrassment, and that 0.06 mg. is the greatest strength which can be borne for half an hour without causing injury. Some individuals who are accustomed to it can, on the contrary, stand a strength of .012 to 0.1 mg. per thousand; Lehmann has also reported some cases which have tolerated a strength as high as 0.20 mg. per thousand.

Recently (1924) Kiichiro Muto has demonstarted experimentally arrest of respiration while the heart continued to beat. The smallest fatal dose has been 1.5% by volume. Electric stimulation shows that the diaphragm reacts. The gas, therefore, acts directly upon the respiratory center and paralyzes it. On the other hand, according to some experts, (Weyl, for example), sulphur dioxide should have beneficial effects owing to its bactericidal power when inhaled for a long period in concentrations which are not too strong. In the opinion of some works managers, the men employed in roasting ores rich in sulphur suffer less sickness, and Floret has noticed a great diminution of tuberculosis among them.

Symptoms

TWENTY parts per million parts of air cause an immediate irritation of the eyes. Amounts greater than this are apt to cause suffocation. The air-passages are highly irritated through the formation of sulphurous acid on their surface. Four hundred parts of sulphur dioxide in one million of air are dangerous to breathe for one hour; this amount "elicits respiratory reflexes. Rarely causes death from edema of the larynx. Trachea and bronchi inflamed. Lung edema is rare." Coughing and sneezing are readily induced. Both men and animals may be habituated to its inhalation. According to Tidy, mice can be brought to tolerate twice the amount they could at first. Air containing from 0.03 to 0.04 gm. per thousand did not affect workmen more severely than amounts from 0.01 to 0.02 gm. affected those unaccustomed to breathing the gas. I found that the majority of guinea pigs exposed for fifteen minutes to one per cent sulphur dioxide were killed. Animals that recovered from this amount died within 24 hours. All of these animals had a severe conjunctivitis. The cornea becomes opaque, and there are dyspnea, cyanosis, and convulsions. Taken internally, there is catarrh of the stomach and chronic sulphuric acid poisoning. According to Kobert, sulphur dioxide has been used for murder only once, and sulphurous acid only once with suicidal intent. The effects on vegetation are greater than on animals. Suits for damages against industrial plants emitting sulphur dioxide for the destruction of trees and other plants are frequent.

Chronic catarrh has been noted following long

exposure. Pedley, on the other hand, found no increased incidence of respiratory disease in Canadian paper mills using the sulphite process, and evidence from other industries, such as smelting, is negative. Kehoe and his associates studied the effect of prolonged exposure to sulphur dioxide in 100 workmen exposed to this gas in the course of their employment. Forty-seven had from four to 12 years' exposure, the severity of which fluctuated through a wide range (70 parts per million by volume) but was still higher in the years before 1927. They found a significantly higher incidence of nasopharyngitis, and of alteration in the sense of smell and taste. The men did not seem more susceptible to ordinary colds, but the average duration of colds was two to three times longer than the average for the control group. They also found a higher rate of abnormal urinary acidity, of dyspnea on exertion, of increased fatigue from work, and of sluggish or hyperactive reflexes, which indicated that sulphur dioxide did not have an effect on the human organism.[7]

Postmortem Appearances

THE blood is very dark, and sometimes has an acid reaction. The hemoglobin is changed first through loss of oxygen, then by decomposition to hematin as with mineral acids. The respiratory tract is inflamed and the lungs are edematous.[7]

Recently I had occasion to examine men employed in a refrigerating plant who had been exposed daily to sulphur dioxide for periods of from two months to 20 years. Inquiry revealed that these men had less colds since working with sulphur dioxide than previous to exposure. Three complained of sinus trouble which was not present prior to their working with this gas. A young man, 26 years of age, who had worked for four and one-half years charging over one-half million units with sulphur dioxide, did not have any disturbances of the eyes, nose or throat. There was no evidence of bronchitis or cardiac disease. Another who had worked for 20 years, with no exposure for the last two years, was found to be in excellent condition, there was no evidence of heart or pulmonary disease. Another employee who had worked with sulphur dioxide for three and one-half years, and who claims he had bronchitis since nine years of age, states that his condition has not been made worse by exposure to this gas. Examination of his chest revealed the presence of a chronic bronchitis.

Rawlins[8] found that in treatment of colds, sulphur dioxide was of no benefit when the cold was of two or three weeks' duration; this seems to be borne out in the forementioned case, as the sulphur dioxide did not benefit his bronchitis. Kehoe and his associates, as previously mentioned, found a significantly higher incidence of nasopharyngitis, both chronic and slight. Two who had enlargement of the thyroid gland, stated that the inhalation of this gas did not affect them. While testing the units for leaks, I noticed that the men who are employed in this type of work did not have the irritation of the throat that I did.

When a container or a pipe-line bursts, spraying the liquid into the eyes, it freezes the eyeball. Clark[9] reported two cases in which such injury occurred .

After the accident the eyelids became swollen, and the cornea and bulbar conjunctiva had the appearance of a hard-boiled egg. There was considerable pain, photophobia, and blepharospasm. Vision was reduced to light perception. After the circulation was restored in the tissues, the treatment consisted of instillations of atropine and dionine along with hot compresses and sedatives. The cornea was denuded of epithelium. The conjunctival surface of the upper lid was transformed into a mass of granulated tissue; this was curetted once and upon several occasions was treated with styptic.

Five months later the left eye showed the following features: The skin and ciliary borders of the eyelids were of normal appearance and texture. The eyeball was extremely sensitive to light and on this account the lids were kept tightly closed. There was some epiphora. Vision was reduced to light perception. Eversion of the eyelids revealed a great amount of scar tissue which resembled trachoma in an advanced stage. The tarsus was not distorted. The bulbar conjunctiva was not particularly altered in appearance except for a rather coarse reticulate vascular congestion. An active scleritis was present in the lower outer quadrant. Ocular rotations were normal. Ocular tension was 22 mm. Hg. (Gradle-Schiotz).

Kennon[10] reported a case of an expulsion of the contents of a tank over the body and face of an employee of a factory making sulphur dioxide.

"The effect was apparently as if hot water had been thrown on him causing injury of the skin of his face, eyelids, and nose. There was then considerable edema of the lids, especially of the right eye, with blistering of the skin of the lids and face. The cornea of the right eye was almost completely denuded of epithelium but the substantia propria was clear. There was marked conjunctival injection and chemosis, and a burn of the conjunctiva adjoining the limbus above and extending upward and inward. In the left eye the injury was not so severe, the epithelium being absent in only the lower half of the cornea. The conjunctival injection in this eye, was less marked than in the right. Both irides were hyperemic and there was considerable purulent discharge from both eyes. Under boric acid wash, atropine and oil instillations, the corneal epithelium was almost replaced at the end of nine days after the accident with vision equal to the counting of fingers."

Four years after the accident this man showed no ill effects whatsoever in regard to the eyes and his general health.

In treatment of frozen eyeballs due to sulphur dioxide, Clark recommends the following: "Wash the eye at once with cold water (ice water preferably) and follow with the application of iced compresses. Local anesthetic and cycloplegic drugs should be employed in sufficient amounts to maintain their therapeutic effect. An antiseptic oint-

ment should be applied between the eyelids. Prompt application of cold water to the injured eye is exceedingly necessary because if this is delayed much greater tissue damage will result."

In several papers recently published, it is claimed that sulphur dioxide caused an attack of asthma. It is stated that between one and two per cent of the inhabitants of the United States have asthma. Bronchial asthma is a form of recurrent paroxysmal dyspnea, characterized by a prolonged expiratory phase, and is an expression, in the patient, of a hereditary predisposition known as atopy. I have not seen a case of asthma in men working over long periods in sulphur dioxide.

Romanoff reported the cases of three electric refrigerator repairmen who developed asthma. Examination of the first case, a well-developed young adult male, showed cyanosis of the lips and fingernails. Respirations were labored. The chest was hyperresonant, and prolonged expiration with sibilant and sonorous rales. The remainder of the physical examination and laboratory findings were negative. The second case had markedly positive reactions to orris root, duck, dog and rabbit epithelium. Intradermal skin tests on the third case were completely negative, except for a slight reaction to egg white.

Frequently the question is asked, can sulphur dioxide cause tuberculosis?

This is best answered from statistics which can be found on page 103 of the Surgeon General's Report for 1920, as follows:

"One hundred seventy-three cases of tuberculosis occurred during 1918 among the 70,552 men who had been gassed in action. Of this number, 78 had been gassed by gas, kind not specified; eight by chlorine; 65 by mustard; and 22 by phosgene. The number of cases of tuberculosis for each 1000 men gassed was 2.45. Since the annual rate of occurrence for tuberculosis among enlisted men serving in Europe in 1918 was 3.50, and in 1919, 4.30 per 1000, it would seem to be apparent that tuberculosis did not occur any more frequently among the soldiers who had been gassed than among those who had not been."

Lt.-Colonel H. L. Gilchrist, M.C., made a very extensive study of this whole subject, including an analysis of over one thousand applications for compensation filed with the Veterans' Bureau.[11] In this report, Colonel Gilchrist concludes that those who apply for compensation a year after discharge claiming to have developed tuberculosis as a result of their army service certainly have no basis for such an assertion. They probably would have become tuberculous whether they served in the army or not. If more than a year elapses from the time of the alleged gassing until symptoms of tuberculosis develop, it is very probable that the gas was not the cause of the disease, provided, of course, that the soldier's lungs had been clear at the time of his discharge.

"In concluding the consideration of these reports concerning the ultimate condition of men who were gassed, it seems quite apparent:

1. That only a very minute percentage have suffered from any permanent disability directly caused by gas.

2. There is not the slightest evidence that gassing has increased the liability to tuberculosis. So far as the evidence goes, men who have been gassed suffer less from tuberculosis than normal individuals under the same circumstances.

3. That neurasthenic conditions are responsible for a considerable part of the disability alleged to result from gassing."[12]

Twedell[13] concluded from his inquiry among managers of sulphur dioxide manufacturing plants, that sulphur dioxide is a preventative if not a curative agent.

Sulphur dioxide has been used in the past as an agent in the treatment of tuberculosis.

Conclusions

SULPHUR dioxide causes an irritation of the respiratory tract from the formation of sulphurous acid, which causes coughing, sneezing and also a conjunctivitis.

2. There is no increased incidence of respiratory disease such as tuberculosis.

3. There is no increase in the number of cases of asthma in men working with sulphur dioxide.

4. No more cases of cardiac disease are found among workers in sulphur dioxide than in those otherwise industrially employed.

5. Liquid sulphur dioxide may cause serious injury to the eyes.

6. Workmen can become habituated to the inhalation of sulphur dioxide.

7. There is no permanent injury from a sudden concentrated dose or from long exposure to small concentrations of this gas. Those exposed to irritating amounts of this gas usually recover and return to work.

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IN CONVENTIONS, this year, all roads lead to Cleveland, O. In June, on days from 5 to 8 inclusive, the Hotel Statler in that city will be the scene of the Twenty-Fourth Annual Meeting of The American Association of Industrial Physicians and Surgeons with The American Conference on Occupational Diseases and Industrial Hygiene. See the Program, pp. 260 *et seq.*

Fractures of the Neck of the Femur

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IT IS with pleasure, and at the same time humility, that I address you on this subject as my interest in it dates back to my association with the late Dr. E. Denegre Martin, a former president of this association.* Through his originality, farsightedness and eminently practical judgment was evolved a method of treatment which today, with no major change, is in essence the favored surgical method for the treatment of this condition by internal fixation irrespective of the fixing agent used. Since his death I have endeavored to carry on his work in this connection, and further experience with it has only served to show its usefulness and indicate its success by the only reliable criterion—i.e., "follow up" of cases over a long period of time.

The present-day popularity of internal fixation dates back only over the past five to eight years, so that the work of Martin begun some 18 or 20 years ago must be given due credit as among the earliest of the present era. Many are the variations in technique, surgical approach, and fixing agents used, but with more widespread acceptance has come the urge for practical simplicity which was the essence of Martin, both the man and his work.

I need scarcely here call attention to the age incidence so well known in this type of case, and yet it is just this problem which, I believe, has been responsible for the establishment of internal fixation on its present-day footing. These patients almost without exception are those individuals in the upper age groups and hence present not only the problems in direct relation to reduction and immobilization of the fracture, but also those incident to the confinement of the aged sick in the recumbent position. Internal fixation has offered what we now feel to be a successful solution to both these problems. By it, the fracture is held immobile at the same time allowing that activity so important to the continued well-being of this type of patient. Age and debility, to my mind and experience to date, have not served as contra-indications to this type of procedure, but rather have been considered deciding factors in urging the adopton of internal fixation. That this attitude is correct is, I believe, proved by the fact that in this series of cases to date, numbering about 80, there has been no immediate operative mortality.

WITH age and debility then considered as indications for this form of treatment, it must be considered in what location the fracture may lie to lend itself to proper maintainance in immobility by internal fixation. Experience to date with these cases prompts the statement that fractures occurring through the neck from the subcapital area to the base at the trochanters are suitable for internal fixation. Intertrochanteric fractures in which the major portion of the great trochanter remains attached to the shaft fragment may likewise be successfully treated in this manner.

The separation of the lesser trochanter from the shaft has not been found to offer any difficulty and causes no concern in the handling of the case. It usually unites by bony union, and has not been the source of any continued disability or interference with function. Fractures of the head proper and those cases in which comminution exists are doubtful subjects for internal fixation, and the decision in such cases must rest with the determination by the surgeon as to whether or not the fixing agent may be placed so as to hold all fragments immobile and at the same time remain permantly immobile itself. A fixing agent not tightly placed so as to be completely immobile had better not be placed at all. In all bone work the fixation or maintainance of position by applied metallic agents requires complete immobility of the agent used—motion or looseness means foreign body.

This, to my mind, is of greater importance than the special metallic content of the material used, and my experience would seem to bear this out.

In the cases which make up the basis for this experience the fixing agent used has been the ordinary hardware carpenter's screw No. 8 to No. 10 usually, with few exceptions, 3 inches in length and "case hardened" for added strength. In their use, now covering a period of 18 years, I have been unable to observe any undesirable effect attributable to the material used.

LET me now consider briefly the pre-operative handling, the technique, and the postoperative course. As for any surgical procedure, hospitalization is necessary and after initial emergency treatment x-ray study must be done for accurate estimation of the position of the fracture line and fragments. (Fig. 1-a.) In these cases there is no urgent call for immediate reduction and operation. In fact, it is contra-indicated, as it only serves to add additional shock and hazard to the picture of a human organism already taxed to nervous and physical capacity by the immediate effects of the injury. A postponement of usually five to 10 days before operation under proper care serves to restore normalcy, or near normalcy, and during this time reposition of fragments has usually been obtained and retained by the use of traction applied according to the Russell method. In some instances it has been necessary to resort to the Leadbetter maneuver to secure reposition of fragments, but this has been the exception rather than the rule, it being possible usually to secure complete reduction by gradual traction alone. Where possible I feel that this is desirable. The technique of application through simple lateral incision has been adequately described elsewhere. Briefly it is as follows: With the patient in position in

* Presented at the Forty-First Annual Meeting of the Association of Surgeons of the Southern Railway, New Orleans, 1938.

Fig. 1-a

Fig. 1-b

Fig. 1-c

Fig. 1-d

Fig. 1-e

FOLLOW-UPS

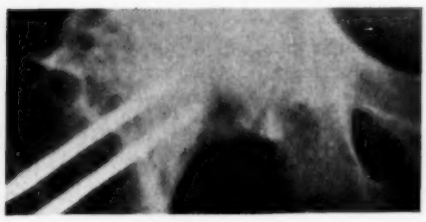

Fig. 1

Fig. 2

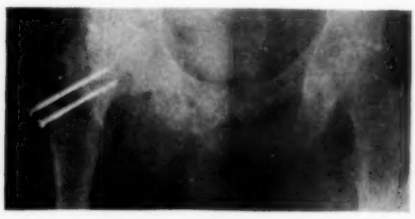

Fig. 3

Fig. 1—Mr. P. eighteen months, postoperative. Operation, December 4, 1929. Fig. 2—Mr. P. five years, four months, postoperative. Fig. 3—Mr. P. eight years, five months, postoperative (Arthritis antedates fracture)

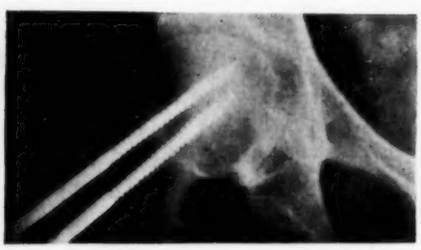

Fig. 4

Fig. 5

Fig. 6

Fig. 4—Mrs. B. eighteen months, postoperative. Operation, February 28, 1929. Fig. 5—Mrs. B. five years, three months, postoperative, or May 21, 1934. Fig. 6—Mrs. B. nine years, three months, postoperative, or May, 18, 1938

Fig. 7

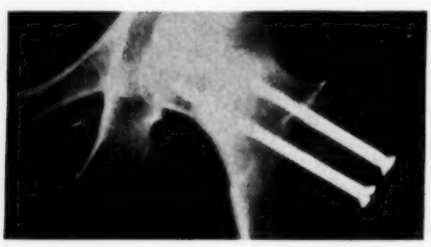

Fig. 8

Fig. 9

Fig. 7—Lucy P. eighteen months. Intertrochanteric fracture, coxa vara but full function. Fig. 8—Mrs. W. eighteen months postoperative. Operation, January, 1932. Fig. 9—Mrs. W. two years, seven months, postoperative. August 9, 1934. (Fracture opposite hip two months ago. Screw applied now to both hips.)

Fig. 10

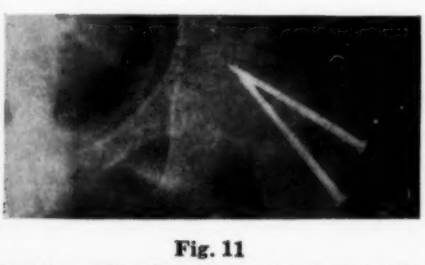

Fig. 11

Fig. 12

Fig. 10—P. A. six months postoperative. Fig. 11—Screw in place three years. (Early case, application of screws, parallel now.) Fig. 12—Fracture eight months old when fixation applied. Plate seven and a half years later

slight abduction and internal rotation to about 30° with traction sufficient to maintain length, portable x-ray is made and developed. (Fig. 1-b.) Simple lateral incision is made with the prominence of the trochanter occupying the upper angle of the incision; soft parts are divided rapidly down to the bone and the subtrochanteric plane exposed.

This is the area of the lateral aspect between the prominence of the trochanter and the shaft, and will usually be seen to be at right angles to the long axis of the neck.

With this fact in mind, and with the first portable plate in view, a point is chosen on the prominence of the anteroposterior curvature from which a drill is introduced approximately to the line of fracture paralleling the long axis of the neck.

It must be remembered that the neck of the bone is normally directed with an anterior inclination of some 10° to 15° from the sagittal plane of the shaft, and the drill must be sufficiently inclined anteriorly to account for this. I am confident that lack of appreciation of this fact has been responsible for failure in one case, for it was formerly thought that the neck of the femur, with the foot and leg held in the position described, would occupy the horizontal plane. It is now known that a slight degree of anterior inclination is necessary. The portable plate is then made confirming direction of the drill, which if not properly placed may be redirected. (See Fig. 1-c.)

The drill is then removed and the first screw inserted but not screwed down—protruding to serve as a guide for introduction of the second screw adjacent and parallel to it. The drill point used is slightly larger than the screw, allowing its free introduction to the depth of the drill hole. In this manner then the screw is set, the threads engage in the head fragment, and as they are tightened the smaller head fragment is pulled to and firmly held against the shaft fragment of the neck. (Fig. 1-d.) The use of two screws increases this action and likewise prevents the possibility of rotation at the fracture site. Closure of soft parts is done with interrupted sutures throughout and final x-ray check, made with lateral view procured by flexing the thigh to a right angle with the plate beneath the buttocks. (Fig. 1-e.)

Postoperatively no plaster or retaining device is used. Mild traction and suspension for a period of one week seems to add a feeling of confidence and security to the patient, and is usually used though not necessary. Sutures are removed after one week, and if post-operative traction has been used it is removed and free motion allowed. The patient is free to assume any desired position, and usually at the end of two weeks postoperative is allowed home, after which he may be up in chair or on crutches if physically able to do so. No restriction as to motion or active use is made except absolute non-weight bearing is insisted upon for a period of six months, or until x-ray shows union.

Local Anesthesia

—in Industrial and General Surgery—

(A Review of Five Types of Local Anesthesia Used by the Author in his Office and Hospital Practice)

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IN THIS paper the author respectfully submits his observations, comment and conclusions relative to the administration of 3320 local anesthetics in 1648 operative procedures. The discrepancy between the number of operative procedures and the number of anesthesias administered arises in the inclusion in this report of 125 hernias, 100 of which were treated by the injection method and 25 operated under general anesthesia, or general and local, or local anesthesia. As indicated in the included summary, 11 operative cases required only 11 anesthetics whereas 100 injected cases required 823 local anesthesias, or 8.23 anesthesias per listed case. Further, our tally has recorded each anesthesia administered in our rectal group of 116 cases. Many of these procedures require two types of anesthesia for one operation and some procedures need be repeated on several occasions; thus we have 373 local anesthesias listed for 116 patients, an average of a little better than 3.2 anesthesias per case.

Truly may it be said that the "Knight of Old" without his trusty sword and steed and shorn of his armor was in no worse fix than is the practitioner of industrial medicine and surgery today without his armamentarium of syringes, needles and stock of local anesthetics. All too frequently he knows little and cares less about the special properties of the more or less toxic substances which he "shoots into" his patient "so that it won't hurt." And yet so rapidly is the never too stable art and science of the practice of medicine moving that the knowledge of ten or even five years ago is, in many instances, outmoded today. With the advent of "Injection Surgery" rapid strides have been made in the science of local anesthesiology, and it needs not my statement here to bring home to the reader that only by constant effort can he hope to keep abreast of the swiftly moving current. Medical schools, as Dr. Waters of the University of Wisconsin Medical School's Department of Anesthesia[1] told the thirty-fifth Annual Congress of Medical Education and Licensure recently, have not yet become conscious of their responsibility to teach their student bodies the fundamentals of this newer branch of the science of anesthesiology, the clinical instruction in which has robbed this writer of his vacation time for the past three years.

FIVE solutions have been found adequate to supply our every need for local anesthesia in our industrial office and hospital practice. These are procaine, quinocaine, butyn, cocaine hydrochloride, and rectocaine. Their relative status in our practice may well apply to the practice of any

industrial or general practitioner as well as to the surgeon. Constantly increasing costs of hospitalization as well as the fear of the hospital and its mysteries present many problems at our door and have resulted in the development of office surgery, less radical, sometimes quite spectacular in the results obtained for the effort expended by the surgeon and the cost incurred to the patient. Truly, the operator has an increased overhead and the patient is often apprehensive, but the cost is included in the patient's statement and apprehensiveness disappears as the operator attains more confidence and knowledge in this branch of his art. It has been our practice, and we strongly advise each practitioner starting in to do "office surgery" under local anesthesia to adopt the same practice, to try *upon ourselves* each anesthetic that we plan to use on others. Much may be learned from this simple expedient, and we have nothing but contempt for the doctor who will administer, routinely, to his patients, a solution which he is afraid to use upon himself.

An anesthetic solution for every purpose! And hang out the shingle imprinted with the unforgettable words engraved thereon: No Pain Here. And why should we in this day and age practice the brutalities of other generations who had not our facilities for the surcease of pain and suffering?

Procaine

The Toxicology of this Drug and Its Consideration in Relation to the Others Comprising, with It, Our Armamentarium Against Pain.

Mayer[2] stated of procaine (novocaine) hydrochloride that this drug should not be used in strength of over 1% and that adrenalin chloride yields bloodless fields, but if used in amounts over 1.0 cc. (1:1000) produces a greater toxemia in combination with either cocaine HCl or procaine; he admonishes never to use adrenalin chloride at all in hyperthyroidism.

Lundy[3] states that procaine hydrochloride is the best and safest of the synthetic local anesthetic agents. Surely it is the most widely used, but we hesitate to state, in an empirical manner, that any local anesthetic is the best in all conditions and situations. That procaine HCl has come in for much abuse because of postoperative pain, especially in dentistry, is no fault of the drug but rather lies in the operator's technique. In its use, as in the use of any local anesthetic to be injected, postoperative pain, sloughing of soft tissues, necrosis of bone as noted following dental extraction, and the all too common and quite painful "dry socket" are all the aftermath of the injection of the solution under pressure. This fact is borne out by our personal experiments with 33 extractions using pressure injections in some and avoiding pressure in the rest. We were able to extract the tooth or teeth without pain to the patient in every instance, but in the 10 where 2% procaine solution and in the four where the standard quinocaine solution was injected under pressure into the spongy "process" severe postoperative pain occurred in every instance. There were six dry sockets and 10 patients extruded particles of necrotic bone ("pieces of process") after from six to 35 days. Of the 19 cases (17 novocaine, 2 quinocaine) where no pressure was used not a single dry socket ensued, and only four extruded necrotic bone, and in each case of the latter the particles of bone were all retrieved within a period of 10 days. This early extrusion leads us to believe that the fragments were broken loose at the time of extraction and did not arise from necrosis resulting from the injection of an anesthetic under pressure, "l.a. pressure necrosis." The author's personal experience was unbearable postoperative pain which was only partly relieved by morphine sulphate in ½ grain doses and the coincident delayed shock and five-day period of absolute prostration following the use of 4.0 cc. and 6.0 cc. of 2% novocaine for nerve block and multiple pressure injections into the process about the 14 teeth to be extracted. My good friend, the dentist, thought he was being "extra-good" to me, and I have yet to convince him that pressure injection is bad. Dead particles of my bony process began to work out after 14 days and the last was dislodged with a pair of tweezers on the forty-fifth postoperative day!

In the use of novocaine we occasionally find patients who do not tolerate the drug in any appreciable amount and it is fairly well known, as Lundy[3] states, that the "intravenous injection of a small dose of the drug is not without danger," convulsions having been brought about in his recorded instance after the intravenous injection of 3.0 cc. of a 1% solution in a patient normally tolerant of an average amount of the drug. We have noted that as little as 1.0 cc. injected inadvertently into the superior hemorrhoidal plexus has caused unconsciousness accompanied by a weak and thready pulse and a drop in systolic blood pressure from 126 to 60 within three minutes of the time the injection occurred.

Case Report: S. H., female, 26. Diagnosis: Acute cryptitis and papillitis, small internal hemorrhoids and proctitis, anal spasm. The treatment was directed primarily toward the relaxation of the anal spasm; 2.0 cc. of 1% novocaine were injected perioneally to form a skin wheal in each quadrant. Ten minutes later 4.5 cc. rectocaine were injected into and around the sphincter ani muscle through each skin wheal and, after another 10 minutes, the sphincter was manually dilated to 4.5 cm. and injection treatment of the hemorrhoids was attempted. Anesthesia was not effective in the inflamed rectal musoca, and the pain was so severe that additional anesthesia in the form of multiple injections of 1% procaine was required before proceeding. At the moment nothing was thought of the fact that the last needle wound bled profusely, but within three minutes the patient had passed through successive stages of dizziness, and drowsiness, and into unconsciousness. On regaining consciousness within a second interval of three minutes the patient was nauseated and vomited but within 30 minutes she had recovered sufficiently to leave the office unattended. This prompt reaction to the drug, in our opinion, came about as the result of its being carried almost instantly and with little opportunity for dilution directly to the liver where, according to Eggleston et al,[4] it is destroyed. The prompt recovery was with the aid of inhalations of aromatic spirits of ammonia only. This probably resulted from the fact that our syringe at the time of injection contained only 1.0 cc. of the 1% solution which the liver

was able to destroy promptly. We no longer use procaine about the rectum except to create a small wheal at the site where a large needle is to be inserted for a more viscid fluid. We also believe that it may be safely used under a fissure, but we avoid it entirely for deep injecting or for injecting into the rectal mucosa. The next "accident" might result in a less fortunate conclusion.

Novocaine is best tolerated in men between the ages of 20 and 50, and in women between 25 and 45, in our recorded experience of 1637 local anesthetics with this drug. We have used it in amounts varying from 0.5 cc. of a 2% solution injected to create a cutaneous wheal under a pigmented mole to 400 cc. of a 0.5% solution in a case of a ruptured gangrenous appendicitis and generalized peritonitis complicating lobar pneumonia. In this latter case the peritoneal cavity was literally washed out with the solution which was expelled from the abdomen as the peritoneum was opened, carrying out with it some 600 to 700 cc. of pus. And with 10 years elapsed since the operation no evidence of intestinal adhesions or obstruction has appeared, although a ventral hernia of the incisional type has been present since operation.

As the years of the patient progress toward old age the toxicity of the drug appears to increase.[3] We have used procaine hydrochloride in patients between the ages of six months and 92 years but at these extremes we prefer to use the 0.5% solution. In our industrial work we find 90% of our cases between the optimum age limits, and have as yet to experience toxic manifestations in this group which approximates 65% of our total recorded local anesthetics.

Perhaps the most satisfying and spectacular results in procaine anesthesia from both our own and the patient's viewpoint lie in the reduction of fractures of the long bones. In this use we get the most complete relaxation of the concurrent protective (?) muscle spasm that can be obtained with any type anesthetic. In addition we obtain the cooperation of the patient which frequently is manifest in active countertraction by the patient whose first positive effort is quite meager, but, as he realizes the complete absence of pain accompanying the effort, rapidly assumes maximum proportions. We have been pulled off balance by the greater effort of the patient on more than one occasion, until now we always get well braced before instructing the patient to exert his counter traction, and even then the author has his more husky associate exert the traction and contents himself with manipulating the fractured parts into position. This use of novocaine solutions in 0.5% to 1.0% strength on our hospital service has received some unfavorable comment on the bases of unnecessary risks of toxic reactions and of introducing piogenic infection into the site of fracture where the traumatized bone has little or no resistance to infection. Our answer to our critics is that toxemia, in the amounts and strength of the solution used, is unlikely unless we should inject directly into a vein, which likelihood is improbable in that if aspiration through the injecting needle into the attached syringe returns thick dark blood we do not inject the solution at that site. Further, injecting as we do only into the site of the fracture and then only with the return of pinkish serum into our aspirating syringe, there will be no "sucking up" of the solution into freshly opened veins for the simple but sound physiological reason that the proximal end of a traumatized vein is quickly sealed by the formation of a soft clot, and it is rare, indeed, that we get a fracture on the operating table for reduction in less than an hour from the time of its occurrence. All except the very largest veins of the extremities tend to seal off their proximal ends in this manner within a period of from three to 15 minutes (excluding varicosed veins in which the normal direction of flow is reversed). We have yet to experience any toxic manifestations in the use of this drug in these cases, nor have we had a single infection in over 75 fractures so treated and of which 47 are included in this report. As one having served under and been taught by Dr. Wm. O'Neill Sherman, whose meticulous technique made bone plating a practical and safe procedure, and whose record of bone work done without contamination or infection is unequaled in the whole history of surgery, we profess to have a most healthy respect for bone and the susceptibility and low resistance of bone to infection, and hence we maintain rigid asepsis in this approach and procedure. And yet, upon a number of occasions, we have witnessed the insertion of an anesthetic needle through dirt and grime without subsequent infection of the injured bone. Perhaps the skin itself — or the spastic muscle through which the needle must pass— wipes it clean of any contaminating piogenes! No matter what may have occurred in a few cases, which likely enough are exceptions to the rule but which serve to prove it in the minds of thinking men, we prefer to be as clean as it is surgically possible to be in the handling of any and all "bone cases" under any and all conditions no matter what type of anesthesia or operative treatment may be used and we do so advise and teach. It is significant that approximately 40% of our fractures in which local anesthesia has been used have been of the open or compound variety and in these we believe that the local anesthetic mitigates against infection because, injected through sound skin to the site of fracture in larger quantities (usually double the amount that would be used in a simple fracture at the same site), the fluid seeks an outlet and thus, surgically clean as it enters, it washes outward in exactly the opposite direction to that through which the infection may have gained its entry at or subsequent to the time of the trauma and escapes through the skin at the site of the skin lesion or opening (which break in the continuity of the skin and/or its underlying structures constitutes the meaning of the terms "open" or "compound" as used in describing the fractures). In addition to this reverse washing out of the wound, we feel that with complete muscular relaxation, the torn and macerated muscle, fascial and tendonous tissues tend to slip out from between the jagged edges of the broken bone—hence their

further traumatism is avoided. An instance of an extreme case of bone trauma and its course is exemplified in the following case history.*

> J.S., male, 49 years old, suffered a crushing trauma to his right forearm when a fall of roof slate pinned the forearm to the top of his cutting machine. We saw the patient about an hour later, after he had been "dug out" by his fellow workmen and transported 10 miles to the Canonsburg General Hospital. Fragments of bone were noted on the external portion of the wound when the first aid dressings were removed. Properly applied tourniquets had controlled the hemorrhage and had also exerted their influence to produce a considerable amount of pressure anesthesia. Analgesia was induced with a "hypo" of ½ grain of morphine sulphate dissolved in 1.0 cc. of 1:1000 adrenalin chloride which latter served to overcome his shock.
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> Small areas of sound skin were cleansed with benzene and ether and painted with tincture of merthiolate, and through these windows in the grease and coal dirt 60.0 cc. of 4.0% novocain were injected widely in a fanned-out manner toward the macerated surface, and after a 10-minute interval a 10-minute scrubbing of the wound with a hand brush and tincture of green soap was carried out, the remaining skin surfaces being cleansed simultaneously. Benzene was necessary for the removal of some of the grease. The wound edges were next widely separated and the resultant cavity filled with the aqueous, 1:1000 solution of merthiolate which was allowed to remain for three minutes, after which an extensive debridement of all devitalized (with considerable vital) tissue was performed. In all, 38 fragments of bone were removed and a defect of more than half of the radius resulted. The remaining portions of the debrided muscles were repaired by suturing their fascial coverings and the skin edges were approximated without drainage through the major wound. A Penrod drain was inserted through a lesser wound, 4.0 cm. long, on the flexor surface after debriding its edges. A plaster splint was applied to the opposite surface from the major wound and extended from the finger tips to the mid-portion of the arm. Healing by primary intention was practically complete in 10 days, and circulation and vitality had so far returned to normal that on the twenty-fifth day Dr. George V. Foster decided that the optimal time had arrived and on May 31, 1935, did a tibial transplant by an onlay graft with screw fixation to fill the radial defect. Healing of the soft tissues again occurred by primary intention and the bone graft attained adequate strength and stability in six weeks for the patient to return to work. With a total morbidity of approximately 70 days—does this not explain our hearty enthusiasm for local anesthesia in fractures!

The novice using any form of local anesthesia will do well to bear in mind that a sufficient time for the anesthesia to "take effect" should elapse between the injection of the drug and the beginning of the operative procedure. We begin our operative procedure after a minimum of five minutes with 2% novocain (probably a longer period should elapse in dental operations); 10 minutes with the 1% solution; and 15 to 20 minutes with the 0.5% solution. The same time, 10 minutes, is permitted to elapse when we use the newer combination of nupercaine and quinine hydrobromide, (Quinocaine, Farnsworth)[5] which we prefer in many instances to novocain for reasons which shall be discussed later in this paper. Ten minutes should elapse in topical applications of 2% butyn or 4% cocaine in the eye, 15 minutes for cocaine in the nose or throat. Fifteen to 25 minutes should elapse after the use of the slowly absorbed oil solution in rectal operations (rectocaine—C. F. Kirk Co.).[6] The operator is always tempted to begin too soon if he remains in the presence of the patient, and one of my former associates gave as a good motto for an overanxious surgeon this admonition: "Go into another room and smoke a cigar. If you don't smoke, this is a good time to learn, and if you get sick it will be for the best interest of your patient who, by the time you have recovered, will have attained a satisfactory stage of anesthesia." After all, the anesthetic is given for the relief of pain, and why should any of us operate before it has become effective. We would not think of operating before our anesthetist flashes on the "go" sign in ether or nitrous oxide and oxygen or any other anesthesia. There is no more occasion for operating "raw" and then having a beautiful postoperative anesthesia when using local anesthesia than there is when using a general anesthetic.

Another important consideration is the need for confidence and a good morale on the part of the patient. This has been brought most forcefully to our attention in the use of local anesthesia by reason of our greater success with the procedure in our office surgery than we find possible to attain in the hospital ward or operating room, even when using considerably larger amounts of the same anesthetic solution in the same strength. At the office, the office nurse, the author's associate and the author are all trained not to say the wrong thing or, in fact, anything at the wrong time. The office conversation takes on the nature of a "verbal anesthesia." All are informed as to what to expect in any situation or any complication arising from the operative procedure or the anesthetic. All instruments are arranged in a cabinet on swinging covered trays within reach of the operator's right hand, and his left hand can reach into a tier of drawers containing every antidotal substance needed for any emergency. We will not use any procedure or local anesthetic without first being conversant with its possible reactions and its specific antidotes or without having the antidote within our reach at every moment. Everything is arranged to prevent confusion and delay, and no instruments are displayed. And then, too, we attempt to build up the confidence of the patient before undertaking any given procedure. Contrary to some practice, it is our plan never to operate at the time of the first visit of the patient if the procedure is an elective one. We like our patient to have time to "talk it over with the family" and suggest contacts with satisfied patients having undergone similar procedures. We also ask them to consider whether any factor exists in their past history which might have a bearing on the present case. These factors, in the given case, are enumerated to the patient, and, having been duly considered and negated, relieve our minds and theirs of the fear of possible accidents or complications.

Let us now consider the situation as of a similar

* This case was presented to the Pittsburgh, Pennsylvania, Surgical Society at its April, 1939, Meeting by Dr. George V. Foster, of the Surgical Staff of the Western Pennsylvania Hospital.

patient—not as an ambulant, office one but rather as one who has chosen hospitalization and has been placed in bed in the institution of his choice to be prepared for an operation. As often, or oftener than not, when the floor nurse appears to take his temperature she finds out that he is to have local anesthesia and forthwith depicts in vivid phraseology her personal fear of local anesthesia, but feels she has redeemed a possible error when on leaving the bedside she coyly remarks: "Oh well, you're brave and I'm not." She hasn't built up his confidence very greatly with her friendly little chat, and should we not use our P.M. and A.M. barbiturates we may expect the patient to have a most restless "time of it" that night. Even with barbiturates, morphia, and other pre-operative medication, we invariably find our patient tense and apprehensive and he often states in an affected offhand manner that he guesses we'd better have the gas machine ready in case "this stuff doesn't work." A thousand strange sounds have been carried to his conscious or sleeping ears during the night. He feels that he's not boss here, whereas had he been in our office he had but to pick up his hat and walk out should such be his desire or should the thought strike him. But here he hasn't even got his pants! A sense of camaraderie pervades the office; our concerns are common; and all are attentive to every phase of "this thing we're going to do." But at the hospital the attendants have a thousand and one other things to do, and the patient hates to bother anyone with his simple wants. And when he gets to the operating room, the patient's morale is weakened further by the dire formality, which at times may be depressing even to the operator and certainly often aggravates the latter to the limit of his endurance by its petty delays! We can do the same operative procedure in the office in one-third the elapsed time it takes to do one in the hospital operating room. Like the medical schools of the country, the hospitals have not as yet found out that the other local anesthetics than procaine are on the market and being widely used. Nor have they found out that ease and smoothness in operating the facilities of their workshop are more essential here than in general anesthesia. Mental shock occurs. A wincing expression upon the face of a supervisor—meant to be sympathetic—can be the source of great harm to an already unstrung and apprehensive patient. What is the answer to this if we are to keep this remunerative minor and major surgery coming to our hospitals, especially our smaller hospitals, which are in need of all the help they can summon to their aid? Let's look ahead! For we in this profession are not greedy and we appreciate the need of our hospitals, as well as our need for them. A special operating room with its crew trained for local anesthetic work, operating smoothly, quietly, without the fanfare of the "big operating room" but with really greater efficiency! A master table, with sterile nurse in charge, with every instrument sterile and no waiting on the part of the surgeon or worrying on the part of the patient! Look out, little hospitals of America! The big city hospitals have the *big stuff* and the *reputation too*, and industry needs to watch its cost sheet closely, and industry's surgeons are keen to feel the pulse of the master and sense the trend of the times! Let the big city hospitals have their big stuff, you'll get your share in any event, but stop feeling that minor surgery, and that major or minor surgery with local anesthesia, are beneath your dignity and not to be given the consideration and careful planning they deserve and demand. In England the office dressing room is dignified with a title, and is known as "The Surgery," and by great leaps and bounds in this country of ours this modest dressing room of yester-year is fast becoming "The Surgery" of tomorrow in fact, as well as in name —and this to the definite financial loss and detriment of the small hospital.

The susceptibility of a patient to pain or the relative absence of such susceptibility has much to do with success or failure of local anesthesia and would appear to influence the toxicity in direct ratio to the degree of pain susceptibility, and it is our opinion that the patient most susceptible "to being hurt" presents a picture of basic nervous imbalance and should not be subjected to this form of anesthesia except for the most trivial procedures, and that even then the more dilute solutions should be used and that the total dosage of the drug should be kept to an absolute minimum. Bad risks of this type frequently point out their weakness and request general anesthesia, and the operator is hereby cautioned to heed the warning when given. Likewise, extreme variations of blood pressure from the average normal, as set up by the American life insurance companies, should contraindicate the use of local anesthesia, as should marked increases in pulse pressure.

BEFORE proceeding to discuss further the use of local anesthesia it may be well to consider the properties desirable in any local anesthetic. These are:

1. Solubility in water or normal saline or in a bland, absorbable oil where prolonged anesthesia or slow absorption is desired.
2. Relative non-toxicity.
3. The solution must permit sterilization in solution without charge in its physical, chemical, anesthetic or toxic properties. In our practice we consider any solution which normally permits of heat sterilization without change of color to be chemically or physically changed if its color varies from normal and we are suspicious of increased toxicity, which would make it dangerous to use. Likewise, a change in color or the presence of flocculation or precipitation due to age or the action of sunlight are considered contra-indications to the use of any solution; this is particularly prone to occur in stock solutions, vials, ampoules or capsules which contain adrenalin chloride. We have demonstrated to each new associate of ours this toxicity of a suspected solution which has changed in color, by injecting 1.0 cc. or less of it and noting symptoms anticipated, i.e., increased pulse rate, dizziness, tinnitus, headache, and nausea or vomit-

ing with occasional cramp-like abdominal pain and shortness of breath.

4. Non-irritability during or subsequent to use. We have noted that the burning sensation ensuing as the effect of the drug wears off follows the use of the stronger solutions or the use of any solution under pressure.

5. Actual and determinable anesthetic value with predeterminable length of time for which anesthetic may be maintained.

6. Compatability with a vaso-constrictor. We consider this optional in that we would rather determine the length of time of anesthesia by the content and amount thereof and depend upon our knowledge of the toxicity of the various solutions and combinations to predetermine which anesthetic is to be used in a given condition.

Quinocaine

THE early use of quinine as a local anesthetic was almost exclusively made by proctologists who seldom ever reported their findings. Combination of nupercaine and quinine probably was suggested first by Dr. E. C. Cook, of Madison, Indiana, who had used this solution extensively. The idea was originated by Dr. Wm. A. Hinckle, of Peoria, who used the hydrochloride salt of quinine. Following Dr. Cook's suggestion, M. G. Farnsworth experimented with different quinine salts, and even though he found little published upon the use of the hydrobromide salt, he found it to be much less toxic experimentally. It was better tolerated and the duration of anesthesia was more prolonged than with the better known urea salts, and its effectiveness was fully equal to any of the other salts. It would appear that there is a definite bond or synergism between this neutral hydrobromide salt and nupercaine which, in Farnsworth's opinion, the empirical use has established beyond question. However, the pharmacological behavior has not at this time been entirely established. The older users of quinine almost always incorporated urea, and this combination, and their combinations, were not well tolerated. As a result quinine also has been almost forgotten as a prolonged local anesthetic. There is little theory to offer, but this is an instance where the results had been determined first, and we will have had to build a theory to fit known and proved clinical facts. The maximum dosage theoretically is 100 cc., and may be administered at one sitting. Routinely 40 to 60 cc. are being used. The nupercaine is conceded to be the only ingredient to be watched, and Ciba[8] states that this ingredient may be given in several times the dosage recommended with quinocaine, the per cubic centimeter content of which is nupercaine 0.00050 gm., quinine hydrobromide 0.00648, normal saline q.s.

"Quinine* possesses marked local anesthetic powers and was at one time considerably used as a substitute for cocaine. For this purpose the hydrochloride of quinine and urea is generally preferred. Because of the insolubility of basic quinine it was formerly believed that the alkaloid was absorbed only from the stomach, but Grosser has shown that even the insoluble preparations are completely absorbed. Hartman and Zila confirm the fact that the insoluble salts of quinine are as well absorbed as the soluble. They also found that if the quinine was given by the mouth the proportion of the alkaloid in the blood remained practically constant for 12 to 24 hours, but that if the drug was injected intravenously 90% of it had disappeared from the blood within 30 minutes. Apparently much of the drug is destroyed in the body, as various investigators have succeeded in recovering only from 40 to 60% of the ingested dose from the excreta. According to Kerner, a portion escapes in an amorphous uncrystallizable form, and a second portion as a substance free from bitter taste, though crystallizable and having the fluorescence of quinine; for this substance he has proposed the name of dihydroxyl-quinine.

"Elimination seems to be freest between the sixth and twelfth hours, and about two-thirds of that which is recovered from the urine is excreted within 24 hours of its ingestion.

"The dose of quinine varies greatly according to the purpose for which it is used as well as the susceptibility of the patient . . . .

Toxicology: even in the quantities employed for therapeutic purposes quinine frequently produces unpleasant symptoms. The most common of these symptoms is ringing in the ears often accompanied with a sensation of fullness in the head, and perhaps some obtruding of the sense of hearing. With the use of larger doses these symptoms are intensified, the deafness is very marked, *disturbed vision may exist,* and the *flushed face,* with the sense of distention in the head, may point towards a cerebral congestion, which is in some cases relieved by spontaneous epistaxis. In decided cinchonism, giddiness and staggering in walking are very common. After toxic doses, severe headache, delirium, *stupor,* complete deafness and blindness, dilated pupils, embarrassment of respiration, great weakness, convulsions, paralysis, and finally collapse, either comatose or delirious, may result. The deafness produced by large doses of quinine usually passes off rapidly; very rarely is there a permanent impairment of hearing. Amaurosis, with a peculiar ischemia of the retinal vessel, has in a small number of cases been produced by very large therapeutic doses of the alkaloid.

"A considerable number of persons have an idiosyncrasy towards quinine and even relatively small doses may produce toxic symptoms. In most of these cases there have been skin eruptions, frequently simulating those of scarlet fever. This type of poisoning is doubtless to be classed among the allergies. It is interesting to note that in many of these patients the sensitivity extends to other laevorotatory alkaloids of the quinine group, such as cinchonidine. Sensitivity towards the dextrorotatory isomers (quinidine and cinchonine) is much less common."

The writer has reviewed approximately 6000 pages of manuscript, text, reprints and clippings and finds much to suggest the anesthetic value of

* From the Dispensatory of the United States, Edition 1937.

such a combination of a quinine salt and nupercaine, but little definite clinical data to support his experimentation. Like Hinckle[7] we have used procaine as the yardstick by which to measure this newer anesthetic combination as well as others. The low content of nupercaine Ciba[8] in quinocaine has given us little fear as to its toxic reactivity in the body.

Early clinical use of the neutral hydrobromide salt of quinine established its toxicity to be the same as the sulphate[9,10] and we have advised the same dosage of 30 grains a day in malaria as of the better known quinine sulphate. Hinckle[7] first used somewhat more toxic quinine hydrochloride with procaine, but this mixture tended to precipitate out upon standing, and he later changed to nupercaine 1:1000 or weaker, and found a definite synergism present in his experience of 250 injections. He reported less postoperative soreness at the site of injection than with other drugs, and in his series no edema, induration, delayed healing or sloughing attributable to the quinine was noted. In our experience with quinocaine we find that doses in excess of 30 cc., administered within an area not greater than 10.0 cm. in diameter, do produce temporary induration and increased vascularization, and we have used such dosage for this definite purpose in our experimentation in the treatment of chronic and acute periarthritis and bursitis.

Our procedure in our treatment of these cases is to inject 30.0 cc. into, above, under and around the offending bursa through a single needle wound with a short beveled two-inch 22-caliber needle. Danger of increasing the intrabursal pressure to a painful or troublesome degree is obviated by the fact that the patient so treated quickly tells you of the pressure, at which point the needle is withdrawn from the sac and the balance of the drug is "laid around" the sac by a process of "fanning" out in a conical area with the bursal sac lying within the largest part of the cavity much as a size-30 dipper of ice cream fits into its cone. We have had uniform success in our limited series of 19 bursal (not counting corns, etc.) cases, and find that from one to three injections are required to effect a symptomatic cure. The gritty sensation of the needle within a bursal sac which is filled with calcium deposit as noted in many old chronic cases has been demonstrably absent upon later insertion of the needle. Our present procedure where there is an effusion into the bursa, as in housemaids' knee and miners' elbow, consists of first injecting the quinocaine solution around the distended bursal sac, and after a 10-minute interval we aspirate the sac with a 17-gauge needle. Then through the same needle, which has not been removed, the sac is filled with a stock solution of sodium morrhuate which in turn is reaspirated after 15 minutes and through the syringe and needle which have been in situ during the interval. Patients treated in this manner disclaim any discomfort after the original needle jab. This induration which we utilize as a source of pressure in bursitis, has also been noted in two of our operative hernial cases in which this solution was used. In each of these cases, coincident with the duration of the induration, anesthesia was maintained for over six weeks.

Fat sloughing was noted in two of our operative cases and the process lasted five and nine weeks respectively. Each patient was badly debilitated and had had his hernia for many years.

Case Report: V. E. C., male, 63 years old, had an "injection" type treatment 35 years earlier which at operation on March 6, 1939 was demonstrated to have fused a portion of the transverse colon to the abdominal wall along the full course of the inguinal canal with a present complete absence of peritoneum of either the gut or the attached parietal peritoneum of the abdominal wall. He had approximately 25 injection treatments within a 12-month period preceding operation and a new indirect inguinal hernia and hydrocele were demonstrably closed when inspected at operation. The hernia of which the patient complained at the time of the operation was in effect no hernia at all but actually was a two-inch adult type—intussusception[11] of the transverse colon occurring by reason of the fixation of this portion of the gut. The intussusception was not constant but occurred as a bulging of the whole inguinal canal with increase of intra-abdominal pressure and with the finger tip following the course of the canal the process of invagination could be felt very definitely, but the diagnosis was missed until visually demonstrated at operation. Repair was difficult but the offending gut was finally transplanted upward about 4.0 cm. and the canal, with its unusually heavy and thick superior and internal margin (the conjoined tendon and the transversalis and internal oblique muscles) was readily repaired and, thereafter, the hydrocele sac was exercised. Extensive fat sloughing subsequently occurred and the drainage continued for nine weeks, but the repair of the canal was never in any danger.

Quinocaine was used here despite a known idiosyncrasy which had been noted as a dermatitis, excessively weeping in nature, which ceased promptly whenever the drug was stopped. We believe the sloughing in this patient's, more or less senile type of low resistant, fat was definitely caused by the quinocaine. Notwithstanding our belief we would repeat the procedure again tomorrow on him because of his excellent condition throughout the whole postoperative period. This we believe in part to have arisen from the profound and prolonged anesthesia which prevented the debilitating pain and discomfort which so often proves to be the postoperative undoing of the patient.

Case Report: W. B. J., male, 73 years old, bilateral non-reducible inguinal hernia, was operated on March 17, 1939, for the incarcerated left inguinal hernia which proved to contain within its two separate yet adherent sacs most of the big gut. Attached to the descending colon were fecal-liths within thin-walled epiploic appendages, each about the size of a hickory nut and numbering a total in excess of 50. These were left discreetly alone and in our opinion were the result of recurrent partial and complete bowel obstructions which had always, although with progressively greater difficulty, spontaneously released themselves. The right hernia spontaneously reduced itself as traction was exerted in releasing the adhesions of the transverse colon from the direct sac in which it was contained. The descending gut was inadvertently opened in releasing a constricted portion and no inflammation of the mucosa in or around the epiploic appendages was noted. This opening was increased somewhat in its length in the line of the long axis of the gut and then repaired in a transverse manner to materially widen the lumen thereof. Exceptionally heavy muscles and fascia were encountered in the repair and these were separated approximately 2.0 cm. and fairly fixed so that approximation for repair was difficult. To overcome this difficulty stainless steel wire sutures[12] were used in an interrupted manner. Upon the first appear-

ance of fluid under the incision (sixth postoperative day) the whole skin and superficial fascial portions of the wound were opened and packed loosely with plain gauze. Healing occurred within 37 days, although granulations on the overlapping skin margins were still present when he left the community on the forty-fifth day.

In each of these cases a total of approximately 50.0 cc. of quinocaine was injected. In younger and more rugged individuals the effect of the drug would have been to cause an induration. In these cases we noted the extensive sloughing of a flabby, already senile, type of fat whose supportive trabeculae of connective tissue seemed to be without healing properties. The drug was undoubtedly the *exciting* cause of the sloughing processes described but its effect was entirely local. The *predetermining* factors were the intereference with circulation because of the bulging pressure of the herniae from within, and in the first case because of truss pressure from without, and the general nutritional and hence trophic deficiencies inherent in each instance because of a general debility, and in the second case because of his repeated partial and temporarily complete obstructions to the bowel, a definite vitamin B_1 deficiency could be demonstrated and was the underlying factor of a right coronary spasm on about the thirtieth postoperative day. In this cardiac accident the exciting factor was the patient's great nervous tension and excitement because of the forthcoming (next morning) speech by one Herr Hitler. Sedation and vitamin B_1 therapy in the amount of 75,000 units cleared up his angina promptly. In absolute fairness to this new anesthetic it should be stated that we have had far more extensive sloughing in hernial repairs of the aged with inhalation anesthesia and with procaine in the 0.5% solution. This was not sloughing of a trophic type in the sense of interference with trophic impulse by reason of the ileo-inguinal and iteo-pubic nerves having been directly traumatized by the injecting needle or by direct action of a concentrated solution of the drug. Our procedure is to lay from 5.0 to 10.0 cc. of the solution around these nerves for a distance of 5.0 cm. from the upper margin of the incision, but no attempt is made nor do we desire to inject directly into the nerve sheath. If such ever were our procedure we might well expect damage to the nerve or its sheath with the use of any anesthetic solution strong enough to effect the anesthesia desired. In our opinion by "laying the solution around" the course of the nerve it is diluted by the tissue fluids and whatever irritating or traumatizing effect it may have possessed originally will be counteracted in part, perhaps neutralized by these tissue fluids.

With but two cases of sloughing in 965 recorded administrations of quinocaine and each of these cases presenting known local and systematic predisposing causes we feel that the statement[13] that "any drug that produces a 24-hour anesthesia or analgesia must seriously depress protoplasmic activity, interfere with the healing of wounds or produce degeneration in the peripheral nerves or roots" is completely in error. This statement would be true if applied to the one-time use of phenol for prolonged anesthesia, the phenol acting by destroying the sensory nerve ends and often the nerve fibres.

Because of its prolonged anesthetic action we refer to this new combination of local anesthetics as our "seven-day anesthesia" although we note that the period elapsing before normal sensation returns is in direct proportion to the amount injected into the area to be treated and thus by adjusting our dosage we may, fairly definitely, control and predetermine the period of anesthesia. In the wide variety of conditions for which we have routinely or experimentally used quinocaine this controllability has given us a great deal of satisfaction and has been the source of a great deal of comfort to our patients, many of whom noted the difference in lasting effect in comparison with that of previous local anesthetics.

The most gratifying results obtained were in those extremely painful conditions which have heretofore given us considerable pause before attempting remedial office surgery. Among these conditions we mention:

Atresia of the cervix.
Suppurative bartholinitis.
Coagulation of infected tonsils for autogenous vaccine effect.
Tonsillar and peritonsillar abscesses.

Intercostal neuritis.
Peripheral neuritis (traumatic).
Injection treatment of herniae.
Operative treatment of herniae.

True hydrocele and hydrocele of the hernial sac.
Varicocele.
Cellulitis in any part of the body.
New growths; hematomata.

Ingrown toenail, nechia, paranechia.
Acute or chronic bursitis.
Fissure in ano, and
External thrombotic hemorrhoids or painful skin tags.

We attempt to follow Danna's[14] idea of air replacement after aspiration in empyema (and suppurative bursitis) and we believe that our lack of universal success in this procedure lies in our inability to determine at the time whether we have succeeded in completely evacuating the purulent content of the thoracic cavity. If we were able to complete this procedure with the patient under the fluoroscope our results would show a much higher percentage of recoveries without subsequent rib resection. Quinocaine, 5.0 cc., is used by infiltration into the intercostal space either at the site to be entered or posterior to it. In the latter event the infiltration anesthesia produces nerve block anesthesia. In our opinion general anesthetics are definitely contra-indicated in aspiration procedures as well as in thoracotomy with, or without, rib resection, and for approximately 12 years we have used local anesthesia, novocaine 0.5% or 1.0% and recently quinocaine with definite lessening of our morbidity and lowering of our mortality rate. We believe that the injection of

air into the thoracic cavity should follow every paracentesis thoracis as a prophylactic measure for adhesions.

At this writing no death has occurred in any of our seven appendectomies, all of which were extremely bad risks. We propose to use for infiltrative anesthesia quinocaine diluted in from equal part to three parts with sterile normal saline solution because of its prolonged action, and we respectfully call this to McCarty's[15] attention as obviating the necessity for haste in gall-bladder surgery under infiltrative anesthesia.

Recently, complicating pregnancy, a thrombophlebitis in a varicosed long saphenous vein, at the level of the knee, was successfully treated by injecting quinocaine around the thrombosed area and applying an elastic adhesive bandage from a point 5.0 cm. below to a point 5.0 cm. above the thrombosis. Pain disappeared immediately and only a fibrous cord remained at the site when the bandage was removed three weeks later, at which time all redness, swelling and tenderness had disappeared. This procedure was suggested by Homan's[16] work.

In the radical operation for ingrown toenail with quinocaine in three recent cases (operation, electrosurgical[17]) our patients experienced no postoperative pain or discomfort, a happy variation from our previous experiences with novocaine and with general anesthesia.

TWO operative accidents of near fatal termination have been experienced in our series of 965 quinocaine anesthetics.

CASE HISTORY: A.M., female, age 40 years. Diagnosis: internal hemorrhoids and cystitis, operated 10-15-38. This patient presented an underlying sympathetico-parasympathetic imbalance which was the result of radium sterilization for menorrhagia and metorrhagia of functional origin. Medical treatment for spastic sigmoiditis over a period of six weeks had been productive of little or no relief of the symptoms, and it was felt that the rectal pathology was an aggravating factor which would have to be removed before she would be relieved.

At operation 18.0 cc. of rectocaine were injected through a 1½-inch gauge needle, through four wheals produced by the previous injection of 8.0 cc. of 1.0% procaine hydrochloride, into and around the quite spastic anal muscle. After a 20-minute interval, the sphincter ani was gently dilated to 4.5 cm. and 5.0 cc. each of quinocaine and a formula "No. 61"[6] was injected around the relatively small, hemorrhoidal masses through approximately 15 needle punctures, three of which bled upon removal of the needle. A running conversation between the patient and the operator had continued during the procedure which required approximately three minutes to complete. As the last injection was completed the patient ceased talking and, when turned over on her back, she seemed to be sleeping quite profoundly, as indeed she was for we were unable to rouse her by slapping, pinching or shouting at her. Within five minutes the patient was snoring deeply, her face was flushed, and her pulse had slowed to 60 and was palpably much weaker than previously. Ten minutes later no radial pulse could be felt, precardial auscultation revealed a rate of 38 and respirations were eight per minute and were shallow and jerky; her skin which had been flushed was pale and covered with great beads of cold perspiration.

Treatment had consisted of 3.0 cc. coramine,[18] 1.0 cc. adrenalin chloride (1:1000), and this was now repeated and in addition 5.0 cc. eschatin[19] was administered by intravenous injection into the right external carotid vein. The head and shoulders had been elevated, blankets applied to conserve body heat, and extremities had been vigorously rubbed. An enormous bowel evacuation (although the patient had had an enema two hours before operation) now occurred and the bladder emptied itself involuntarily. In 10 minutes a third 1.0 cc. of adrenalin and 3.0 cc. of coramine were administered and 35 minutes after onset of the coma the patient drowsily inquired if the operator was through. By this time the respiratory rate and amplitude were normal and the pulse rate was throbbing through at 80 per minute. Another hour elapsed before the patient could walk wobbily out of the office.

No further treatment has been required for either the spastic sigmoiditis or rectal pathology, neither of which caused the patient any further trouble.

The immediate postoperative course was uneventful except for an "awful weakness in my legs and a thumping headache for a week" thereafter.

CASE HISTORY: Inguinal hernia No. 49. M.R.C., male, age 31 years. Diagnosis: right direct, complete, reducible inguinal hernia; duration, eight years — associated pathology, undescended and undeveloped right testis.

The patient had had eight injection treatments with apparent closure of the hernia (6-24-38 to 8-13-38); 22,000 units of antuitrin S[20] had been administered during the same period with descent and development of the testis to a normal position and size. Quinocaine and neoplasmoid[6] had been used to inject the hernial area. An hydrocele developed during the descent of the testicle and has been successfully treated with injections of quinocaine and 5% sodium morrhuate.

After three months a "sneaker" (small indirect inguinal hernia resulting from failure to obtain closure of the congenital hernial sac) appeared and during the fourth injection treatment our second "accident" occurred. Considerable induration of the deep tissues was present, and the needle point was placed with some difficulty at its desired position on the floor of the canal just mesial to the vascular structures. Aspiration produced no blood, but after 3.0 cc. of the mixture of quinocaine and neoplasmoid had been injected the plunger suddenly "fell away" from the pressure-exerting thumb and from 0.5 to 1.0 cc. of the mixture entered the vein. The syringe action was immediately reversed and 10.0 cc. of blood aspirated; the patient was told what had happened and asked to watch for symptoms of the effect of the drug, which symptoms were enumerated. Eschatin and coramine were loaded into syringes and within three minutes were needed.

The first symptom noted by the patient was a tingling, then a burning sensation of the skin over his whole body. The skin assumed a brick-red appearance. He next stated that he felt "funny, dizzy," and drowsy and involuntarily turned on his left side and went promptly off to sleep. Within five minutes of the time he received the solution into his circulation his respirations were down to five, his radial pulse was gone and the precardial rate per minute was 30, his skin was becoming pale, and he was beginning to perspire. His breathing was further impeded by a thick, muco-purulent, post-nasal discharge which dropped into his throat. Within 15 minutes the patient received 7.5 cc. of coramine and an equal amount of eschatin and at that juncture was given 1.5 cc. of adrenalin chloride. The throat had been aspirated by the suction machine and this procedure was necessary for an hour. The head of the table had been elevated to 60°; and blankets had been applied, for the skin had become cold and drenched with profuse perspiration. He had voided what seemed to be an enormous quantity.

After 30 minutes the pulse, which had dropped as low as 15 per minute, could be barely palpated at the wrist and was running between 36 and 40. Respira-

tions were eight per minute and he was "pouring out" great quantities of perspiration. This condition persisted for 75 minutes in all before he was finally and apparently easily, partially revived with inhalations of aromatic spirits of ammonia. Help was then called and, after he had emptied his stomach of 2800 cc. of what appeared to be milk, he was walked up and down a long hallway for another 30 minutes. During this period he had three copious stools and otherwise his condition most resembled that of a narcotic addict who had gotten too large a dose. He constantly begged to be let alone and stated that he "wanted to get some sleep."

After two hours had elapsed he was bundled up and taken to the hospital where two liters of 10% glucose in normal saline were administered intravenously. Mental confusion had persisted but now quickly cleared up, and he dropped into a normal sleep from which he awakened early the next morning. He dressed himself and came out into the hall apparently normal in every way, and "got tired of waiting" for me and left the hospital. No after-effects were experienced, and he returned to work (on the farm) as soon as he got home.

The toxic manifestations in each of these cases were directly attributable to quinine for the most part.[3,9,21] Keyes and McLellan[22] quote Uhlman[23] to the effect that nupercaine (quinocaine constituent) is five times as toxic as cocaine, and go on to point out that the least toxic solution for local work is a 1:2000 solution which is the strength of the nupercaine content in quinocaine. They point out that death has ensued in doses as low as 900 mg., while patients have survived 1500 mg. That nupercaine would seem to have an affinity for the central nervous system appears to be the conclusion derived from the number of instances of paralysis following its use as for caudal or for spinal anesthesia. This fact may explain the great depth of the coma in my cases, but in the main the symptoms were those of quinine poisoning. The first patient had never taken quinine; did not know how it might affect her. The second case had had, in all, 11 previous injections but none intravenous as was this. I have used quinocaine experimentally in 5.0 cc. doses intravenously in 10 instances in varicosities of the legs with no untoward effect except in one instance of a 63 year old "railroad man" who noted a "faintness" which in truth was a mild coma lasting about five minutes and accompanied by drenching perspiration. In the instance of varicosities of the leg, it must be noted that we usually have a retrograde venous flow which permits of greater dilution and slower absorption than does a direct return flow which probably arrives at the liver within two seconds of the time of injection and in almost full concentration.

Much time and space have been spent on the discussion of quinocaine to the end that its qualities might be better known and with a desire to excite an interest in the use of the drug by presenting some unusual procedures, and also for the purpose of preventing "accidents" in the use of this excellent combination. Our conclusion is that, while it is an excellent anesthetic with a controllable duration of anesthesia, it should not be injected purposely into the circulation, and every effort should be made to prevent the accidental introduction of the solution into the venous blood stream. This is true, in our opinion and as indicated by our observations in the instance of all local anesthetics. They belong in the tissues to be anesthetized and not in the blood stream; and an old teaching is to the effect that quinine should never be used in any form or manner wherein or whereby it is rapidly absorbed. The liver is unable to eliminate it rapidly and the drug "piles up" in the system.

A moderate toxicity has been noted in 13 instances in which the patient has a light chill occurring four to six hours after the injection and will feel as if he is "taking down with the grippe" for the next 24 hours. This is also a quinine reaction, and occurs occasionally after the oral injection of the drug.

In our opinion coramine is the antidote of choice when a severe quinine reaction occurs. We disagree[18] with Schube who feels coramine acts as a direct chemical-physical antagonist to barbiturates and further combats the affinity of the body for that drug. Both the barbiturates and quinine depend for their disposal in the body upon the liver. Our personal concept of the action of coramine is to increase the oxygen content of the red blood cells and tissues generally by increasing the respiratory rate and excursion. We like to consider it, in our office and home practice, as our emergency oxygen tent. In its action we seek counter effect to the depressing action of the offending drug plus an increased rate of oxidation and destruction, hence excretion, of the offender.

The stimulation of all vital functions is best accomplished by adrenalin but, when large doses are used, we must consider the possibility of the delayed action of the drug which may produce extreme elevations of the systolic blood pressure and possible cerebral accident. The cortical substance eschatin, apparently combats shock as effectively and larger doses may be used and no present or delayed rise in blood pressure need be anticipated. Its action is somewhat slower, we are told, but in use we have observed traumatic cases, in whom no radial or precardial pulsation could be obtained, respond before the injecting needle could be withdrawn (from the carotid vein).

In our opinion the initial dosage of coramine in acute quinine poisoning should be 6.0 to 10.0 cc., of eschatin 5.0 cc. Both drugs may be repeated and the same or lesser dosage administered in 15 minutes if necessary. May we again impress upon the reader the absolute necessity of having these drugs available in adequate quantities should a local anesthetic emergency occur. These emergencies pop up out of an apparently clear sky and a patient can succumb while you helplessly wait for the druggist to deliver the necessary drugs.

The possible effectiveness of the aromatic spirits of ammonia in reflexly stimulating respiratory rate and excursion should not be overlooked in any systemic reaction to local anesthesia.

Butyn

BUTYN is a non-irritating, easily sterilized, prompt and profoundly acting drug which usually is without unsatisfactory after effects. Its toxicity is, in our experience relatively high and

we have confined its use to a 2% solution which we have used topically in the eye for removal of foreign bodies and debridement of lacerations of the sclera and conjunctiva and their subsequent repair in 204 instances with eminently satisfactory anesthetic effect. Topically in the vagina, the aural canal, the urethra and the rectum we have found it to be of little value.

When first introduced into the eye it is somewhat irritating, but as anesthesia occurs promptly this is of little import.

A 5 or 10% spray may be used in the nose, to replace 10-20% cocaine hydrochloride. We have never seen any idiosyncrasies for this drug and it should be noted that, in itself, it is not antiseptic in its action.

As earlier stated we use it in a 2% solution and in an ointment base containing 1:3000 bichloride of mercury.

Mayer[2] states that neither butyn solution nor cocaine solution should be injected submucously or intradermally, and that cocaine solutions should not be injected into the recently traumatized urethra.

Cocaine Hydrochloride

THERE is a marked difference in tolerance of individuals for this drug. No drug in the pharmacopeia with the possible exception of belladonna displays as wide a variance in the dosage tolerated per kilo of body weight as does cocaine.

We are more familiar with the toxic symptoms of this drug which are: shortness of breath, increased pulse rate, sleeplessness, an increased ability for work but a decreased efficiency, a variable period of excitement, a notable tremor, or a sense of tremulousness as expressed by the patient.

Convulsions result from its excessive stimulation. This period of stimulation is followed, if the dose is overwhelming for the particular individual, by marked depression of the vital functions ending in a cardiac inhibition and finally, paralysis.

The antidote for cocaine poisoning is pre-operative barbiturate medication as a preventive medication. We use[24] sodium amytal in doses of one grain for each 10 kilos of weight, or if the nervous constitution of the patient is such that the sodium salt is an excitant we advise the plain[24] amytal or phenobarbitol in doses of one grain for each 20 kilos of weight. Intravenous solutions should be within arm's reach for use in marked excitement or suspected collapse.

As will be seen in the "Summary," we use comparatively little cocaine. For conjunctival anesthesia we use a 4% solution of the HCL salt in 1:3000 solution of bichloride of mercury, which is the anesthetic solution prescribed for the eye by the chief surgeon of the U. S. Steel Corp., Wm. O'Neill Sherman, in our manual of uniform procedures. Our eye surgeon in the Pittsburgh area (U. S. Steel Corp.) Charles F. Kutscher, admonishes against the use of ointment eye anesthetics as inimical to subsequent treatment should such be necessary. In our private practice we find the 2% solution of butyn to be less irritating upon introduction and more lasting in effect, and if the condition is such as to demand continuous anesthesia we use 1 or 2% butyn ointment[25] which also contains bichloride of mercury in a strength of 1:3000.

For the occasional nasal case requiring an anesthesia we find the 4% solution to be most satisfactory and in traumatic cases upon inserting the expanding type of nasal splint, Simpson's intra-nasal tampons,[32] we find that the application of 10 or 15 drops of this solution to the outer end of the sponge-like splint before saturating it with normal saline gives our patients much comfort and rapidly shrinks the engorged turbinates. In this latter use we have no fear of toxicity because of the washing away of the solution by the increased mucous secretion induced by the trauma and presence of the splint.

Rectocaine

RECTOCAINE[6] contains three powerful anesthetics in oil of sesame. It is readily absorbed into the tissues and even has a value as a surface anesthetic. If a drop or two is rubbed into the lip, it becomes numb within a short time.

Although rectocaine was not placed on the market until the early part of 1936, the experience with the preparation dates to some time previous. A physician in New York specializing in proctology had been using considerable quantities of the Gabriel[26] type solution but was dissatisfied on account of occasional sloughing and apparent inconsistent results. The result of his and the C. F. Kirk Co.'s investigation was the present formula which differs from the older ones in that the anesthesin of Gabriel's solution and the ethyl para-amino-benzoate described by Morgan[27] have been replaced by propyl-amino-benzoate. This same physician has used more than 1000 ampules of this new preparation and he states that he has not to date experienced a single slough following its use. He finds no toxicity. The solution is well tolerated when injected to the amounts 10 to 15 cc., and he finds that there is prolonged anesthesia averaging about two weeks.

Like the older preparations this newer one contains benzyl alcohol and procaine base, the properties of which as an anesthetic are fairly well known. Procaine base is soluble in oil and the oil used is the oil of sesame. This replaces the oil of almonds used in Gabriel's and other similar and earlier preparations. The rapid action of procaine is slowed down by the oil which is slowly absorbed into the tissue surrounding the point of injection.

While both procaine base (special (?) type called Kirkaine base) and benzyl alcohol have definite anesthetic value, most reliance is placed on the propyl-amino-benzoate. This anesthetic is the one which makes rectocaine essentially different from all others in common use. Propyl-amino-benzoate was first mentioned in literature about 1908. Reference to it appears in the United States Dispensatory: "The internal dose is approximately one to four grains, although Cluger asserts that

Totals of Tally Record to Demonstrate the Author's Experience with Five Types of Local Anesthesia in Various Operative Procedure as Classified in his "Office Summary"

(After a Brief and Unsatisfactory Experience with Each of Several Other Types of Local Anesthesia these were Discarded in Favor of the Five Listed Below)

Group	Classification	Type of Local Anesthesia							
		Procaine			QUINOCAINE (Farnsworth) (Nupercaine (Ciba) Quinine Hydrobromide)	Butyn 2%	Cocaine Local 4%	RECTO-CAINE (Kirk)	Total
		½	1%	2%					
I. Gynecological	1. Conization and/or coagulation		5	4	9				18
	2. New growths			1					1
	3. Incision and drainage vaginal abscess			1					1
	4. Uretheral carbuncle		1	2			1*		4
	5. Uretheral poiypus—benign			1					1
	6. Imperforate hymen			1		1 topical			2
	7. Cervical dilatation for acquired atresia		2	2	3				7
	8. Injection treatment—separation of pubis				5				5
	9. Biopsy of cervix			2					2
	10. Pelvic cellulitis and peritonitis—Colpotomy		4						4
	11. Suppurative:								
	(a) Bartholinitis—Coagulation to produce a large patulous artificial duct				2				2
	(b) Incision and drainage		10						10
	12. Br. Lig. Inj. tr. for retroversion				6				6
II. Operative procedures about the head and spine	1. Scalp			1					1
	2. Eyes:								
	(a) Foreign bodies					187	211		398
	(b) Laceration of sclera or conjunctiva or cornea		1			17	57		75
	3. Ears		1	2	3		5		11
	4. Nose		1	2			34		37
	5. Face		2	10	11		1		24
	6. Throat:								
	Coagulation of tonsils for:								
	(a) Autogenous vaccine effect		10	89	51		34		184
	(b) Incision and drainage for tonsillar abscess			7	4				11
	(c) Incision and drainage peritonsillar abscess			7	9				16
	(d) Retro-pharyngeal abscess:								
	(1) Lateral approach*			2					2
	(2) Pharyngeal approach**			1					1
	(e) Uvula-amputation of elongated						6		6
	7. Teeth—single or multiple extraction			27	6				33
	8. New growths:								
	(a) Soft palate				1				1
	(b) Jaw—Benign epulis			1					1
	9. Lumbar puncture		8	2	4				14
III. Neck	1. Cervical adenitis			9	2				11
	2. Thyro-glossal cyst.:								
	(a) Operative excision of			1					1
	(b) Inj. tr. with sclerosing agent			4	2				6
	3. Extrinsic CA. of larynx ligation of anterior carotid artery for hemorrhage		1						1
	4. Exophthalmic goitre-injection treatment				5				5
	5. Removal of neuro-sarcoma arising in brachial plexus		1						1
	6. Regrowths of neuro-sarcomo arising in brachial plexus		1						1
			1						1
	7. Biopsy of regrowth of neuro-sarcoma arising in brachial plexus				1				1
IV. Chest	1. Diagnostic paracentesis:								
	(a) Thoracis			20	3				23
	(b) Abdominalis			33	18				51
	2. Ear drum—Paracentesis					11	14		25
	3. Thoracotomy:								
	(a) Without rib recestion		2						2
	(b) Rib resection		4						4
	(c) Tunnel for:								
	(1) Extra peritoneal subphrenic abscess				1				1
	(2) Peritoneal subphrenic abscess		1		2				3
	4. Pleurisy with effusion-aspiration		8						8
	5. Pneumothorax		8						8
	6. Suppurative mastitis		3						3
	7. Intercostal neuritis			2	1				3
V. Abdomen	1. Appendicitis:								
	(a) Complicating diabetes	1							1
	(b) Complicating influenza	1							1
	(c) Complicating bronchial pneumonia	1	1						2
	(d) Complicating lobar pneumonia		3						3
	2. Secondary repair		5						5
	3. Subphrenic abscess—abdominal route (rib margin)		1						1
	4. Pyloroplasty—phagedenic perforated ulcer of pylorus		2						2
	5. Gall stone and subperitoneal abscess following cholecystotomy		1						1
	6. Carcinomatosis abdominalis		1						1
VI. Hernia	1. Inguinal—injection treatment		107	269	290				666
	2. Femoral—injection treatment				47				47
	3. Umbilical—injection treatment			9	14				23
	4. Ventral—injection treatment			33	49				82
	5. Rare—Petit's triangle—injection treatmen'				5				5
	6. Operated—Canonsburg Gen. Hosp.:								
	(a) Inguinal				7				7
	(b) Femoral		2						2
	(c) Ventral (incarcerated)		2						2
	7. Hydrocele of hernia sac				25				25

TOTALS OF TALLY RECORD TO DEMONSTRATE THE AUTHOR'S EXPERIENCE WITH FIVE TYPES OF LOCAL ANESTHESIA IN VARIOUS OPERATIVE PROCEDURE AS CLASSIFIED IN HIS "OFFICE SUMMARY"

(AFTER A BRIEF AND UNSATISFACTORY EXPERIENCE WITH EACH OF SEVERAL OTHER TYPES OF LOCAL ANESTHESIA THESE WERE DISCARDED IN FAVOR OF THE FIVE LISTED BELOW)

GROUP	CLASSIFICATION	TYPE OF LOCAL ANESTHESIA							
		Procaine			QUINOCAINE (Farnsworth) (Nupercaine (Ciba) Quinine Hydrobromide)	Butyn 2%	Cocaine Local 4%	RECTO-CAINE (Kirk)	Total
		½	1%	2%					
VII. Male genitalia	1. R. hydrocele (Tunica vaginalis)—injection treatment		2	15	52				69
	2. Varicocele				19				19
	3. Dorsal slit		3						3
	4. Circumcision	1	1						2
	5. Hypospadias (dilatation of cicatricial contraction of abnormal opening of urethra)					1 topical			1
	6. Epidydimitis			1					1
	7. Abscesses originating in genito-urinary tract:								
	(a) Perineal		1						1
	(b) Pararectal		1						1
	(c) Prostatic		1						1
VIII. Extremities	1. Soft parts:								
	(a) Axillary adenitis—suppurative		23						23
	(b) Inguinal and femoral adenitis — suppurative		16						16
	(c) Cellulitis		114	33	14				161
	(d) New growths—single or multiple excision and biopsy	15		26	13				54
	(e) Lacerations		16	5	1				22
	(f) Varicose veins				18				18
	(g) Foreign body—removal of		19	14	3				36
	(h) Neuritis; paraneural infiltrations				3				3
	(i) Hematoma:								
	(1) Incision and evacuation of	1	41	20	18				80
	(2) Under finger or toe nails		52	37	5				94
	(j) Finger:								
	(1) Paranechia		21	68	13		1 topical		102
	(2) Nechia		35	16	13				65
	(3) Periostitis and/or osteitis of terminal phalanx of a finger	16	3		3				22
	(4) Perforating wound, bullet:								
	Right popliteal space		1						1
	Left foot		1						1
	2. Bones and Joints:								
	Fractures	2	43	2					47
	Luxations		2	1					3
	Malpositions		2						2
	Adhesions, interarticular				3			4	7
	Osteitis				1				1
	Nail removed (ingrown)			6	3				9
	New growth (bony spur)		2	1					3
	Amputation of macerated fingers (all types)			17					17
	Removal of bone plate		1						1
	Coccygodynia—injection treatments				5				5
	3. Bursae:								
	(a) Olecranon:								
	(1) Excised				1				1
	(2) Injection treatment		5		8				13
	(b) Subdeltoid				17				17
	(c) Sub-acromial				17				17
	(d) Sub-scapular				2				2
	(e) Ischeo-gluteal			1	1				2
	(f) Pre-patellar:								
	(1) Injection		3		9				12
	(2) Operation—excised		1		1				2
	(g) Corns and callous and warty growths		63		11				74
	(h) Scalenius anticus syndrome				3				3
IX. Rectal	1. Hemorrhoids:								
	(a) Internal	2	22	9	37	1 topical		34	105
	(b) External	2	19	17	33			27	98
	2. Fissure	1	10	6	21			8	46
	3. Fistula	1	3		1			4	9
	4. Cryptitis and papillitis	2	13	8	15			23	61
	5. Pararectal abscesses				2			3	5
	6. Spasm and hypertrophy of the levator ani muscles		1		3			4	8
	7. Pilo-nidal sinus or cyst		11	3					14
	8. Ano-spasm (retracted anus)		10					10	20
	9. Adeno-carcinoma of rectum							1	1
	10. Rectal prolapse following trauma due to ingestion of glass particles		1					1	2
	11. Atresia		2					2	4
	Totals	46	765	851	955	218	364	121	3320

* Out cases—Died 10 days later of bronchial pneumonia. 2nd case—Died on operating table of spontaneous rupture of abscess and drowned in her own pus.

** Died 27 months later of rheumatic heart disease which originated with tonsillitis and cervical adenitis preceding the reto-pharyngeal abscess.

15 grains may be given without harmful symptoms." Sollmann,[4] in his manual of Pharmacology, mentions a dose of four to eight grains. It is evident, therefore, that this is a relatively non-toxic drug although it is a powerful anesthetic. It is much more soluble in oil than is benzocaine, which is a common constituent of some other similar preparations.

Oil of sesame was selected as a solvent due to the fact that the manufacturers have been using this oil as a base for injections over a period of more than 20 years, and have been able to obtain a highly refined product which in itself produces no irritation and is readily absorbed.

Gabriel[26] in 1929 described the use of 3% anesthesin with 5% benzyl alcohol dissolved in olive oil, and had excellent results in pruritis ani and anal fissures. His technique has been changed but

little in the 10 years since his paper was published. He described a slight burning sensation after injection, which is less common in the newer preparations containing the rapidly acting procaine base.

Gorsch[28] in 1934 added the use of the skin wheal of 1% novocaine (procaine hydrochloride) to make the procedure, of injecting the heavier oily solutions, less painful. He further pointed out that if the procaine hydrochloride mixed with the oily solution the result caused pain. In our use of rectocaine we have noted this result is an intense aching pain which lasts several hours but does not affect the end result.

Morgan[27] in 1935 describes the use of proctocaine which contained butyl-para-amino-benzoate and the procaine base with benzyl alcohol, and states that it is the property of the latter ingredient to facilitate the gradual diffusion of the combination.

In our practice between 350 and 400 (2000 cc.) ampules of rectocaine have been used upon 60 patients presenting 127 pathological conditions. In addition we have used the preparation in four cases of joint adhesions.

In our rectal cases we have seen no sloughing but continue to be careful not to inject the solution immediately under the skin or mucous membrane where it might "pool" and cause a later slough. The author caused considerable urinary difficulty when he, in injecting himself to relieve anal spasm and prolapsus recti following the ingestion of broken glass (in candy supposedly containing ground nuts), inadvertently, and by reason of the awkwardness and difficulty incident to operating upon himself, injected about 2.0 cc. of the solution into the prostate gland.

For the past two years the literature has been full of mention and discussion of these oil soluble anesthetics and description of operative procedures used therewith.[28,29,30] We have found that the use of rectocaine often obviates the necessity for operative interference. In fissures rectocaine is used for relaxation but normal saline remains the solution of our choice for underlaying the fissure proper. Contrary to the opinion of some writers we find the long-lasting qualities of the drug a godsend in most fistula and para-rectal abscess cases. General or caudal block anesthesia is used primarily and after the operative procedure is completed we then inject the oil into the sphincter area (occasionally one levator muscle requires injection also), and our patient doesn't have *any* postoperative pain. This injection has made the use of the "rectal plug," gauge packs and rubber tubing passé in operative hemorrhoid work. If used alone in fistulas and abscesses (we did this once) the pain of injection and for many hours thereafter is so intense as to be almost unbearable. It is felt that too much distortion exists, pre-operatively, for any degree of accuracy to be obtained, and we have therefore changed our procedure as indicated.

In post-traumatic inter-articular adhesions and joint adhesions complicating chronic bursitis quinocaine is first injected into the periarticular structures and 10.0 cc. of rectocaine is then injected into the joint cavity. The induction coil is placed over the offending joint and set at from 8 to 15 M.A. for 15 to 30 minutes, the M.A. being determined by the patient's response. The adhesions are then broken up by manipulation. Some degree of pain always accompanies the manipulation, but this subsides in a few minutes. The oil in the solution seems to prevent reforming of the adhesions and is absorbed in about two weeks, during which time the patient is able to move the member freely and after which, in our four cases, the condition required no further treatment.

No toxic reaction has been noted in the use of rectocaine in our experience, or has such a reaction been noted in the literature or by fellow practitioners whom we have interrogated.

When using rectocaine extensive electro-surgery in the form of coagulation or the use of the asector is avoided on account of the danger of the coagulum extending deeper than we intend because of the anesthesia, the inability of congested tissues to carry away or dissipate the heat through the circulation, and the fact that the oil solution, itself, tends to hold the heat and retard normal dissipation.

COMMENT: We have found no contra-indication, in our experience, to the combined use of any two or more of our five anesthetics, provided that they not be mixed; for example, 2% procaine for a skin wheal, quinocaine for periarticular injection, and rectocaine for inter-articular injection. However, none of the aqueous solutions was or should be mixed with the other or with the oily solution.

One case report of a death occurring within two minutes of the injection of 30.0 cc. of 1% procaine into the neck has been withheld because of lack of pathological verification; autopsy was refused. The solution used presented a definite light amber color and the age and strength of the solution were never accurately confirmed. Death was by respiratory paralysis, with the patient having quickly expressed a sense of suffocation. This was our only (and unconfirmed) anesthetic death.

Conclusions

FIVE local anesthetics suffice to constitute an adequate armamentarium for either hospital or office surgery.

2. The use of local anesthesia and the knowledge of technique and toxicology have advanced and are advancing by leaps and bounds.

3. Certain definite properties and the knowledge of proper dosage and toxicity of the various anesthetics discussed provide a basis of evaluation and a definite electivity for the various drugs.

4. Errors in judgment of proper dosage and accidents in or during the operative procedure of injection of local anesthetics may constitute a hazard to the life of a patient.

5. Idiosyncrasies to any drug may exist. This is true of any drug used for the purpose of producing local anesthesia.

6. The operator must be conversant with and

have at hand for immediate use all known antidotes for the drugs he is using.

7. Hospitals will do well to develop and encourage the proper use of proper local anesthetics.

The author would be remiss if he failed to acknowledge the help, the cooperation and the constructive argumentation, suggestions and criticisms of his associates. The loyalty and the efforts expended in maintaining adequate records by GEORGE D. PATTON, M.D., Mayo Foundation, Rochester, Minn., JOHN A. NAVE, M.D., Beaver Falls, Pennsylvania, and EUGENE W. HODGSON, M.D., Houston, Pennsylvania, have made this paper possible, while the accurate classification of operative procedures and the tedious and difficult tabulations of the anesthetics used, by Hannah Townsend, R.N., have been essential to a proper presentation of the subject matter.

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Southern Railway Surgeons

THE Forty-Second Annual Meeting of the ASSOCIATION OF SURGEONS OF THE SOUTHERN RAILWAY SYSTEM will be held at the Willard Hotel, Washington, D. C., on June 13, 14, and 15, 1939. The Southern Railway's Chief Surgeon, DR. GLENN I. JONES, and the District of Columbia members of the Association will be convention hosts. The Arrangements Committee includes DRS. A. R. SHANDS, J. F. MITCHELL, L. S. GREENE, W. B. MASON, H. A. FOWLER, and J. H. LYONS, all of Washington. The Ladies' Auxiliary is in charge of MRS. THOS. H. HANCOCK, of Atlanta, Georgia, its President.

The Program Committee, including DR. F. WEBB GRIFFITH, Asheville, N. C. (Chairman), DR. T. B. REEVES, Greenville, S. C., and DR. B. S. LESTER, Birmingham, Alabama, has prepared the following:

TUESDAY MORNING

June 13, 1939

8:00—10:00—Registration.

10:00—Meeting called to order by DR. JAMES F. MITCHELL.
Invocation: REVEREND DUNCAN FRASER.
Address: Commissioner MELVIN HAZEN.
Address: Assistant Surgeon-General W. F. DRAPER.
Response: DR. EDWARD T. NEWELL.
President's Address: DR. W. H. FRAMPTON.

TUESDAY AFTERNOON

2:00—Address: MR. S. R. PRINCE, General Counsel Southern Railway System, Washington, D. C.

1. "Treatment of Fractures of the Clavicle," DR. W. C. BOSTIC, SR., Forest City, North Carolina. To open discussion: DR. A. P. MCELROY, Union, South Carolina.

2. "Miscellaneous Use of Metal Fixation in Bone Surgery," DR. O. L. MILLER, Charlotte, North Carolina. To open discussion: DR. SAMUEL O. BLACK, Spartanburg, South Carolina.

3. "Skull Fractures and Brain Injuries," DR. W. H. BLAKE, Sheffield, Alabama. To open discussion: DR. W. W. HARPER, Selma, Alabama.

4. "Principles in the Treatment of Fractures," (with Lantern Slides). DR. E. DUNBAR NEWELL, Chattanooga, Tennessee. To open discussion: DR. W. S. NASH, Knoxville, Tennessee.

5. "Chronic Bone Infections. Report of Cases. Slides, Specimen." DR. EDWARD T. NEWELL, Chattanooga, Tennessee. To open discussion: DR. A. R. SHANDS, Washington, D. C.

6. Showing of Film: "Low Back Disorders." DR. W. W. HARPER, Selma, Alabama.

WEDNESDAY MORNING

9:30—7. "Treatment of Fractures of the Shaft of the Femur." DR. MUIR BRADBURN, New Orleans, Louisiana. To open the discussion: DR. L. D. BOONE, Aiken, South Carolina.

8. "Traumatic Surgery of Upper Extremities," DR. J. B. JOHNSTON, St. George, South Carolina. To open the discussion: DR. R. N. HARDEN, Greensboro, North Carolina.

9. "Conservatism in Traumatic Surgery," DR. JAMES F. MITCHELL, Washington, D. C. To open the discussion: DR. B. W. MCKENZIE, Salisbury, North Carolina.

10. "Abdominal Conditions as the Result of Trauma," Dr. William P. Herbert, Asheville, North Carolina. To open the discussion: Dr. H. L. Brockman, High Point, North Carolina.

11. "The Kidney in Traumatic Surgery," Dr. H. A. Fowler, Washington, D. C. To open the discussion: Dr. Tom R. Barry, Knoxville, Tennessee.

12. "Treatment of Burns," Dr. J. D. Martin, Jr., Atlanta, Georgia. To open the discussion: Dr. B. S. Lester, Birmington, Alabama.

13. "Healing of Wounds—Past and Present," Dr. John A. Hunnicutt, Jr., Athens, Georgia. To open the discussion: Dr. P. W. Boyd, Winchester, Virginia.

WEDNESDAY AFTERNOON

2:00—14. "Some Suggestions on Traumatic Surgery, Especially the handling of Minor Cases," Dr. W. H. Anderson, Booneville, Mississippi. To open the discussion: Dr. E. Howe Miller, Danville, Virginia.

15. "Abdominal Injuries," Dr. A. deT. Valk, Winston-Salem, North Carolina. To open the discussion: Dr. W. R. Rogers, Bristol, Virginia-Tennessee.

16. "Compound Fractures, their Management," Dr. Wm. A. Boyd, Columbia, South Carolina. To open the discussion: Dr. T. B. Reeves, Greenville, South Carolina.

17. "Fractures of the Forearm," Dr. W. S. Nash, Knoxville, Tennessee. To open the discussion: Dr. S. R. Miller, Knoxville, Tennessee.

18. "The Prophylaxis and Treatment of Tetanus," Dr. J. R. Shacklette, Jeffersontown, Kentucky. To open the discussion: Dr. W. S. Austin, Knoxville, Tennessee.

19. Round Table Discussion.

THURSDAY MORNING

9:30—20. "What Should Organized Medicine do about the Medical Indigent," Dr. Albert C. Jackson, Jasper, Alabama.

21. "Our Contributions to Socialized Medicine," Dr. J. B. Shoun, Elizabethton, Tennessee.

22. "Socialized Medicine," Dr. F. Webb Griffith, Asheville, North Carolina. Discussion on the above papers opened by: Dr. W. W. Harper, Selma, Alabama. Dr. J. W. Vernon (Alternate) Morganton, North Carolina.

Unfinished Business. Election of Officers. Selection of 1940 Meeting Place.

New Books

Review by

I. S. Trostler, M.D., F.A.C.R., F.A.C.P.,
Chicago

ROENTGEN DIAGNOSIS OF THE EXTREMITIES AND SPINE, with 508 roentgen ray studies and five clinical illustrations; 435 pages: bound in gray cloth with brown back. By A. B. Ferguson, M.D., Director of Roentgenology, New York Orthopedic Hospital; Instructor in Orthopedic Surgery, College of Physicians and Surgeons, Columbia University. Price $12.00.

This is Volume 17 of the Annals of Roentgenology and is a worthy addition to a series of fine monographic atlases.

Instead of describing the roentgenographic findings in the usual anatomic terminology, this author adheres to clinical diagnostic descriptions, and by so doing produces a graphic mental picture of the subjects of his descriptions, in the mind of the reader.

Chapters on Calcareous Matter; Reticular Bone; Diaphyseal Fractures and Infections; Diaphyseal Malignancy; Metaphyseal Bone; Inherent Disturbances of Bone Formation; Cartilage Lines; Fracture Lines and Anomalies; Fractures; Healing of Fractures; Epiphyses and Small Bone Masses; Non-osseous Tissues; Arthritides; Tuberculous Arthritis; Non-tuberculous Infectious Arthritis; the Spine, etc., make up a most complete and comprehensive text.

The illustrations are well selected and finely reproduced. The arrangement of the subjects is in logical sequence and presents a most pleasing, instructive and orderly systemization.

Case histories are presented at the ends of many of the chapters, so that the reader feels as though he were attending a dry clinic upon the subject. This feature of itself adds materially to the value and usefulness of the book.

The make up, format, printing of the text and illustrations and the comprehensive indexing are excellent.

All in all, this fine book is an important contribution to the growing literature of roentgenology and orthopedic surgery, and a valuable addition to any medical or surgical book shelf.

Saturated Atmosphere

—In the Treatment of Injuries—

Murray B. Ferderber, M.D.,
Research Fellow in Fever Therapy,
Department of Industrial Hygiene,
School of Medicine, University of Pittsburgh

HEAT has been used therapeutically in many forms for hundreds of years including such modalities as packs, steam baths, electrical applications, such as short wave, diathermy, etc.*[1] Any or all forms are used to produce the same effect, i.e., increased circulation of the part involved. Some means are considerably safer than others combining simplicity, flexibility, and control of conditions.

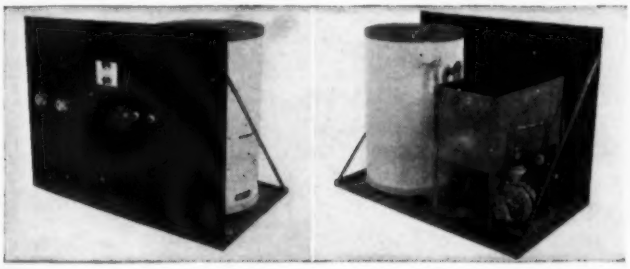
Hyperpyrexia (fever therapy, hpyerthermia, etc.) has become an accepted form of treatment that has been employed quite successfully where its uses are indicated.[2] However, there are numerous patients who require some form of heat therapy but who object to general treatment where only one extremity may be involved. Then, too, there are patients who have reached the age or who are prematurely physically unsuited for general therapy. These patients require treatment of the local part to relieve them of their symptoms, which frequently are quite severe. To this end, the Department of Industrial Hygiene has devised an apparatus which has produced eminently satisfactory results and is now being used in hospitals affiliated with the University.

Physiologically, heat produces dilatation of blood vessels which in turn increases the circulating volume of the part involved.[3] Capillaries which are normally very small become engorged with blood, the elements of which are instrumental in producing relief of symptoms and in many cases complete resolution of the pathological processes. Because of the stimulated circulation, the affected area receives blood supply capable of handling the metabolic processes and relieving symptoms which frequently are severe and are caused by the accumulation of metabolites. While studies in blood volume have not been carried out completely, it

* From the Department of Industrial Hygiene, School of Medicine, University of Pittsburgh.

would appear that those principles which hold that the circulating volume increases in general fever[4] should also hold for heating any part or parts of the human body. From a previous report of this Department,[2] we have shown that the polymorphonuclear leucocyte increase with its phagocytic action aids in cleaning up certain processes in the involved area.[5]

The apparatus used is a prototype of the fever therapy device of Houghten, Ferderber, and Gutberlet.[6] Its present form was adapted by the same workers and consists of a cylinder 43 inches long and 16 inches in diameter, one end of which is open (Fig. 1). Within this apparatus, at its closed end, is a duct and spray so devised that in using the hot and cold water systems of any building, the proper mixtures of these will cause a stream of humidified air within the cylinder at the temperature desired. Evidence from work produced in this Department and the basic work of the American Society of Heating and Ventilating Engineers have definitely shown that saturated atmosphere will produce an increase in temperature and is, therefore, more desirable because the danger of burns is greatly lessened on account of low dry bulb temperatures.



This water spray moves a column of saturated air at a rate of 400 feet/minute and the gradient is no greater than 1°F. This apparatus requires 20 to 30 gallons of water per hour but after the optimum temperature of the enclosure has been reached, the volume of water used is reduced by one-half.

In most industrial hospitals the whirlpool and foot baths are used for soaking and heating an injured extremity. The temperature of these baths usually ranges from 108 to 120° F. and most frequently the lower range is used because discomfort becomes too great at the high temperatures. Since this apparatus uses humidified air and since the heat transfer is not as great, we are able to carry these temperatures at a higher range without any discomfort and definitely without danger of burning. To verify this, we placed one extremity within the apparatus enclosure and the other in a water bath and raised the temperatures of both simultaneously. When a temperature between 112 to 115°F. was reached, the foot was withdrawn from the water bath because of discomfort, but the leg treated in the described apparatus remained and no complaints of discomfort were elicited.[7]

In addition to the routine physical examination the skin temperatures of affected parts were measured using a portable potentiometer indicator with an automatic reference junction compensator with a range of 25-125°F. and a limit of error of 0.5°F. The thermocouple for skin surface temperatures consisted of an iron constantan thermocouple in an aluminum handle with a limit of error of 0.75°F. between 90-110°F. (Fig. 2). Pulse rates were taken every 10 minutes during treatment and while the average increase was only nine beats and the maximum 25 beats, Fig. 3 would indicate that the changes are not too great. The white blood counts were taken with a Bureau of Standards pipette and counted in a Levy-Hausser counting chamber. The average increase of all counts amounted to approximately 1500 cells, the greatest increase was 3800, and there was one slight decrease (Fig. 4). The slight rise in body temperature is insignificant and the increase in pulse rate is usually found in the first treatment due, perhaps, to psychic stimulation and frequently there is a definite drop because this treatment inflicts no apparent hardship on the cardiovascular system. It is interesting to note that men tolerate temperatures as high as 125°F. comfortably, while women

are most comfortable between 115-118°F. Old age and debility are not contra-indications nor are hypertension or cardiac disease. Since the affected parts rest on a sponge mattress and since the apparatus is used on a regular hospital bed, the patient is comfortable, restful, and may either read or frequently fall asleep. We have observed that the improvement period following each treatment is considerably longer than the preceding one until complete relief from symptoms was noticed in the majority of cases. Pain in the affected part was not increased irrespective of the length of time, but this does not hold true in the use of certain other methods of heating.

We have treated many patients, industrial and otherwise, who have sustained fractures following which there has been stiffness of the joints and tendons caused both by fixation and by presumably non-specific inflammation. These patients were treated with ordinary measures but little or no improvement resulted. Following preliminary studies treatment was begun, each period varying in length from one to three hours. Improvement was noticeable from the beginning and since the first treatments of a course were given, daily, progress was rapid and very satisfactory. In rare cases, gentle manipulation was performed, and the progress of these patients was considerably enhanced. There were additional cases where open reduction was performed and where pitting edema, indicated by the shiny, stretched skin, resulted.

In using local heat the stimulation produced increased drainage and a restoration of function and size of the limb so that convalescence and hospitalization were simultaneously reduced. Sprains, either simple or complicated, constitute a very common injury in industrial practice. Due to pathological changes in and about the ligaments, pain and swelling so impede motion that "time off" may run into rather amazing figures. We have observed that although a simple sprain may recover with no treatment, the time required becomes so excessively long that we felt this method of treatment would be even superior to common procedures followed in industrial practice. To our extreme satisfaction results were uniformly good, and relief of symptoms and reduction of edema were noticeable from the beginning of treatment.

The author has treated several cases of varicose and indolent ulcers of the leg in the past several months using iontophoresis[8] and some undesirable actions have taken place. The apparent danger of imminent collapse with absorption of the drug mitigated against this treatment where age and poor circulatory systems were present. Upon the advice of a colleague we treated a few varicose and indolent ulcers with excellent results. One patient in particular deserves mention in that a skin graft on the site of an indolent ulcer appeared to be sloughing and intensive treatment daily was given resulting in the graft "taking" with no further complications. This same patient has been seen frequently since, and there has been no breaking down of the operation described. Occasionally, following crushing injuries, osteomyelitis with sinus formation and drainage becomes a serious complication. Since most of these become chronic, draining sinuses are troublesome to the patient and to the surgeon. Local treatments with surgical procedures have in many cases stimulated the part to repair to the extent that healthy granulation tissue and often cessation of drainage have followed. We do not feel that this constitutes a recognized treatment for this ailment, but it aids in supplying a form of heat that seems efficacious.

In the past several years attention has been drawn to the treatment of peripheral vascular diseases.[9] Those individuals with arteriosclerosis, but who are otherwise fit, constitute a great number of cases we have seen. In the Physical Therapy Department of one of the local hospitals, passive vascular exercise[10] has been used extensively as a method of treatment. While we feel that this method is helpful, we are undergoing the experience of treating many such cases with the method described. The appearance of "blotches" in the extremities treated would indicate that extensive hyperemia had resulted; but these same areas disappeared when the temperature of the part returned to normal. This apparatus might prove extremely valuable in peripheral vascular disease since these principles still hold true for the production of hyperemia.[11] If the blood supply to this part can be augmented by the engorgement of collateral vessels, then an adequate supply of blood could be made available, since the volume of the narrowed sclerosed arteries is insufficient. Although this report is not intended as a resume of our cases, we feel safe in stating that the results obtained justify its use extensively. The increase in skin temperatures coincides with the relief in symptoms and the increased activity without pain permitted the patient indicates that there is much virtue in its use. In a paper to be published in the future by this Department, complete details will be given of this experimental work.

Every industrial physician attempts to prevent injuries; but should they occur, treatment is started immediately to rehabilitate the worker and to prevent complications. Frequently the attempt to rush treatment may carry with it the danger of recurrence or complications; yet a few additional hours of intensive therapy might have prevented any further exaggeration of the injury. Since this method requires from two to three hours for each treatment, it is obvious that the injured worker can not devote much time to his occupation. However, since most other procedures require from 10 to 30 minutes for each treatment, the total number of treatments necessary for recovery is considerably greater. We feel, therefore, that a few treatments of longer duration should produce a better clinical result than many short ones. In the hospital of an industrial plant in this vicinity this moist heat apparatus has been installed to permit the attending physicians to evaluate its use. The reports are uniformly encouraging despite the fact that the time required for each treatment is considerably longer than any other device used for this purpose.

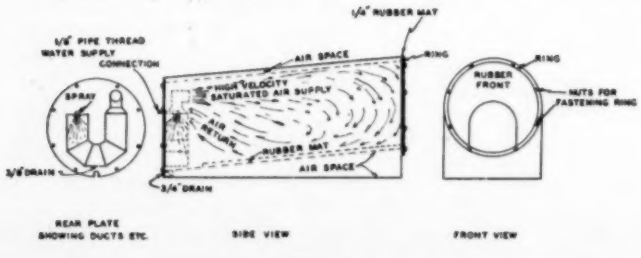


Fig. 1
Apparatus for Producing Saturated Atmospheres

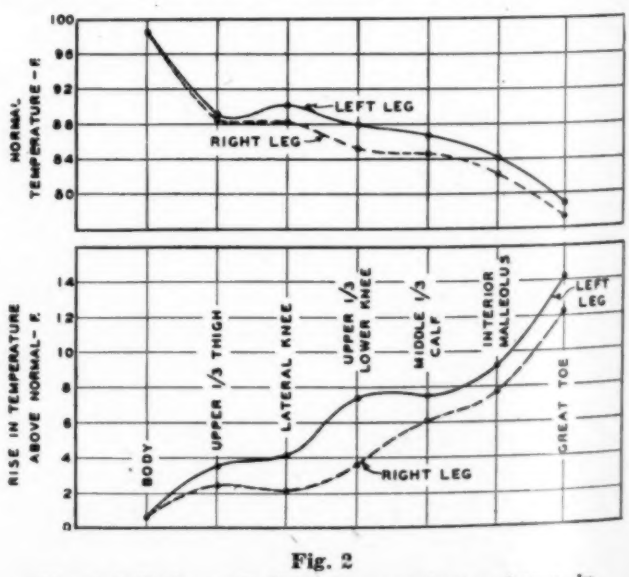


Fig. 2
Normal Body and Skin Temperatures shown in Upper Section. Lower Section shows Rise in Body and Skin Temperatures above Normal after exposure to 115° to 120°F. Saturated Air

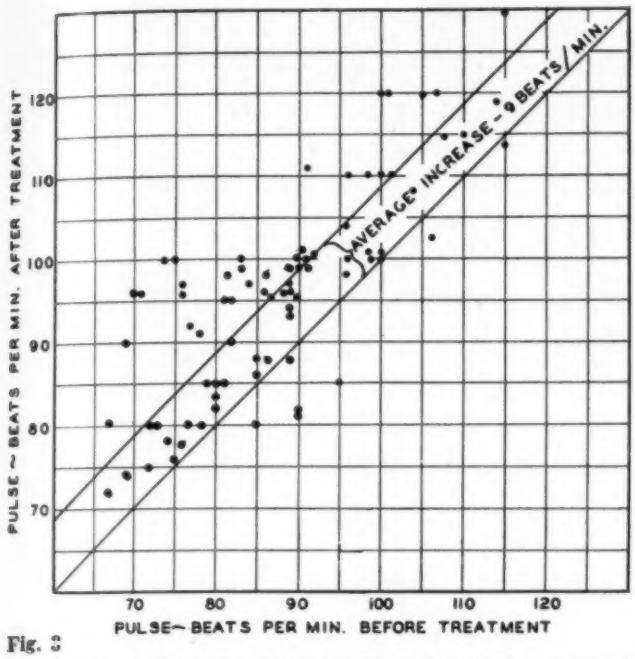


Fig. 3

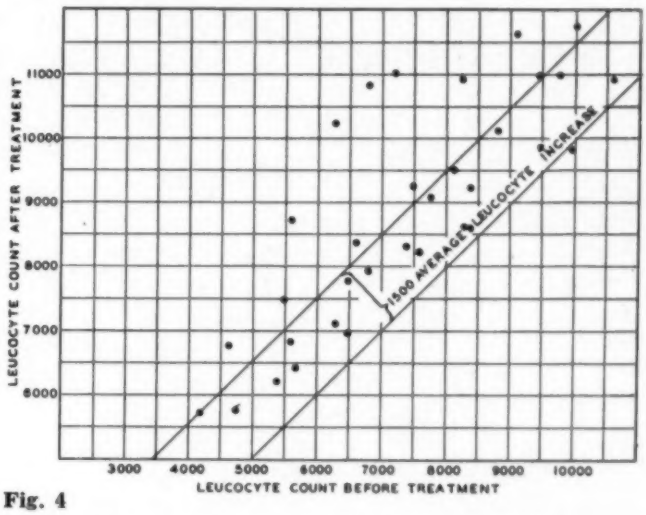


Fig. 4

FIG. 3—Relation between Pulse Beats per minute before and after Treatment. Lower Line indicates No Change in Pulse. Upper Line indicates Average for All Points. Difference between Two Lines represents Average Increase in Pulse. FIG. 4—Relation between Leucocyte Count before and after Treatment. Lower Line indicates no change in Leucocyte Count. Upper Line indicates Average for All Points. Difference between the Two Lines represents Average Increase in Leucocyte Count

With the danger of electric shocks and burns eliminated, constant attention is not needed, and the burden on attending nurses is lessened.

In a recent paper on "Fango Treatment of Arthritis,"[11] it appears that low dry bulb saturated heat is the desirable condition sought by the authors. Since the constituents of the substance used do not have any specific therapeutic value, it would appear that the saturated heat is the medium through which improvement of the arthritic is obtained. The apparatus which we use answers these desirable qualifications and since the temperature can be controlled, and since ease and simplicity of handling are obvious, it would appear that this simple mechanical device might prove helpful in ameliorating painful symptoms.

Conclusion

WE HAVE attempted to show in this report that the type of heat used in the treatment of injuries is very important, and the rapidity of recovery frequently depends on the measures taken. The vast increases in skin temperatures, especially in portions of the feet, where circulation is none too good, would indicate that the vascular system has been stimulated. The slight changes in pulse rates do not contra-indicate its use in the patient with an impaired cardiovascular system. With freedom from burns and shocks virtually eliminated, a greater degree of safety and efficacy has been obtained. We have attempted to show that the experiences gained in both the general and industrial hospital would indicate that this type of heat can be used and might prove extremely valuable in the treatment of certain types of injuries in the general field of industrial medicine.*

* This investigation was made possible by a grant from the Westinghouse Electric & Manufacturing Company to the Department of Industrial Hygiene, School of Medicine, University of Pittsburgh.

[The author wishes to thank DR. T. LYLE HAZLETT, Director of this Department, for his patience and his encouragement to the author in carrying out this experimental work. He acknowledges also the cooperation and clinical help extended by DRS. W. O. SHERMAN, U. HUBER WAGNER, A. W. SHERRILL, SAMUEL SHERMAN, and the other members of the St. Francis Hospital staff. Finally the author wishes to thank DR. F. C. HOUGHTEN, Research Director and MR. CARL GUTBERLET, Research Assistant, of the American Society of Heating and Ventilating Engineers, as well as MRS. CHARLES H. SCHWERIN, who have given their time and efforts in making this publication possible.]

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American Association of Industrial Physicians and Surgeons Twenty-Fourth Annual Meeting : with The American Conference on Occupational Diseases and Industrial Hygiene

FOLLOWING is the Complete Program of the Twenty-Fourth Annual Meeting of the AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS with the AMERICAN CONFERENCE ON OCCUPATIONAL DISEASES AND INDUSTRIAL HYGIENE, Hotel Statler, Cleveland, Ohio, June 5, 6, 7, and 8, 1939:

MONDAY MORNING
June 5, 1939

8:00-9:30 Registration.

W. P. YANT and C. D. SELBY,
Chairmen of the Session

9:30 Opening of AMERICAN CONFERENCE ON OCCUPATIONAL DISEASES AND INDUSTRIAL HYGIENE and 24th Annual Meeting of the AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS.

Address of Welcome: HON. HAROLD H. BURTON, Mayor, City of Cleveland.

Response for the Association: C. D. SELBY, M.D., President, American Association of Industrial Physicians and Surgeons.

10:00 "Health of Workers," R. R. SAYERS, M.D., Chief, Division of Industrial Hygiene, U. S. Public Health Service, Washington, D.C.

10:30-11:00 INTERMISSION: Inspection of Scientific and Commercial Exhibits.

11:00 "Physiologic Effects of Hot Atmospheres," F. C. HOUGHTON, Director of Research Laboratory, American Society of Heating and Ventilating Engineers.

12:00-2:00 LUNCHEON.

MONDAY AFTERNOON

2:00 "The Public Health Aspects of Occupational Hygiene," HENRY F. VAUGHAN, DR.P.H., Director, Public Health Commissioner, Detroit Department of Health.

2:30-3:00 INTERMISSION: Inspection of Scientific and Commercial Exhibits.

3:00 "Fatigue in Industry," D. B. DILL, Harvard Fatigue Laboratory. (Lantern Slide and Motion Picture Demonstration.)

MONDAY EVENING

6:30 DINNER: Guest Speaker, WHITING WILLIAMS, Analyst of Public Opinion, Cleveland, Ohio, who will speak on "The Public Significance of Industrial Health Programs."

TUESDAY MORNING
June 6, 1939

WARREN A. COOK
Chairman of the Session

8:30-8:45 "Use of Interferometer for Two Component Mixtures," G. C. HARROLD and L. E. GORDON, Industrial Hygiene Laboratories, Chrysler Corporation.

8:50-9:05 "Modern Dust Control in Underground Mines," D. E. CUMMINGS, Director of Field Activities, Saranac Laboratories.

9:10-9:30 "Evaluation of Benzol Exposure," H. B. ELKINS, Chief Chemist, Bureau of Occupational Hygiene, Massachusetts Dept. of Labor and Industry.

9:35-9:55 "Study of Motion Picture Industry in Detroit," W. G. FREDRICK, Bureau of Industrial Hygiene, Detroit Department of Health.

10:00-10:20 "A Study of Silicosis in the Silica Brick Industry," W. B. FULTON and A. E. DOOLEY, Bureau of Industrial Hygiene, Pennsylvania Department of Health.

10:25-10:40 "Apparatus and Procedure for Vapor Toxicity Studies," D. D. IRISH, Biochemical Laboratory, Dow Chemical Company.

10:45-11:00 "Possibilities of Control of Lead Exposure by Examining less-than-24-hour Lead in Urine Samples," E. C. BARNES, Industrial Engineer, Medical Department, Westinghouse Electric and Mfg. Company.

11:05-11:25 "Exposure to Fluorine in Industry," W. F. MACHLE, Kettering Laboratory of Applied Physiology, University of Cincinnati.

11:30-11:50 "Lead Absorption versus Lead Poisoning," R. A. KEHOE, Kettering Laboratory of Applied Physiology, University of Cincinnati.

11:55-12:05 "Toxicity Studies on Some Cadmium Compounds," H. T. WALWORTH, Bureau of Industrial Hygiene, Detroit Dept. of Health.

12:05-1:30 LUNCHEON.

TUESDAY AFTERNOON

W. P. YANT,
Chairman of the Session

1:30-1:50 "Toxicity of Benzols," H. H. SCHRENCK, Chief Chemist, Health Division, U. S. Bureau of Mines.

1:55-2:20 "Vapor Tension Method for the Determination of Solvents in Air," L. SILVERMAN and G. REESE, Department of Industrial Hygiene, Harvard School of Public Health.

2:25-2:45 "A New Method of Counting Impinger Samples," C. R. WILLIAMS, Petrographic Engineer, Liberty Mutual Insurance Company.

2:50-3:10 "Calibration and Use of the Gas Interferometer," F. A. PATTY, Director of Industrial Hygiene, Fidelity & Casualty Company of New York.

3:15-3:30 "Air Sampling of Asbestos Dust—Comparison of Impinger and Electrostatic Precipitator Methods," J. W. FEHNEL, Industrial Hygiene Laboratory, Metropolitan Life Insurance Company.

3:30-4:00 INTERMISSION: Inspection of scientific and commercial exhibits.

4:00 Organization meeting of the American Conference on Occupational Diseases and Industrial Hygiene.

TUESDAY EVENING

6:30 NELA PARK—BUFFET DINNER
Demonstration of lighting research facilities of the General Electric Lighting Institute. "Visibility and Comfort in Seeing," M. LUCKEISH and F. Moss, General Electric Company, Cleveland.

WEDNESDAY MORNING

June 7, 1939

G. F. SYKES, M.D.,
Medical Director,
White Motor Company, Cleveland,
Chairman of the Session

9:00 Opening of the Twenty-fourth Annual Meeting of the AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS.

9:30 "The Maintenance of Mental Health in Industry," F. J. ROETHLISBERGER, Graduate School of Business Administration, Harvard University, Boston, Massachusetts.

10:00 Discussion.

10:15 "Fatigue and Employment," R. R. JONES, M.D., U. S. Public Health Service, Washington, D.C.

10:45 Discussion.

11:00 INTERMISSION: Inspection of scientific and commercial exhibits.

11:20 "Plastic Surgery in Relation to Industrial Injuries," DONALD GLOVER, M.D., Associate Clinical Professor of Surgery, Western Reserve University Medical School Director of Surgery, St. Luke's Hospital, Cleveland.

11:50 Discussion. 12:05 LUNCHEON.

WEDNESDAY AFTERNOON

H. L. DAVIS, M.D.,
Chairman of the Session

1:30 "The Problem of Medical Service for the Small Industrial Plant," M. N. NEWQUIST, M.D., Assistant Director, American College of Surgeons, Chicago; Medical Director, The Texas Company, New York City.

2:00 Discussion.

2:20 "The Relationship of the Industrial Medical Department to other Personnel Activities," H. W. LAWRENCE, M.D., Personnel Director, Fisher Body Cleveland Division, General Motors Corporation.

2:50 Discussion.

3:10 INTERMISSION: Inspection of scientific and commercial exhibits.

3:30 "Geriatrics in Relation to Employment," W. A. SAWYER, M.D., Medical Director, Eastman Kodak Company, Rochester, New York.

4:00 Discussion.

WEDNESDAY EVENING

6:30 ANNUAL BANQUET. Toastmaster: C. D. SELBY, M.D., President, American Association of Industrial Physicians and Surgeons. Guest Speaker: WILLIAM S. KNUDSEN, President, General Motors Corporation, Detroit, who will speak on "Human Relations in Industry," after which he will present the KNUDSEN AWARD for the most outstanding contribution to Industrial Medicine, 1938-39.

THURSDAY MORNING

June 8, 1939

R. C. ENGEL, M.D.,
Chief Surgeon,
Cleveland Division, Republic Steel Corporation,
Chairman of the Session

9:00 "The Repair of Tendon and Nerve Injuries," SUMNER L. KOCH, M.D., Chicago, Illinois.

9:30 Discussion.

9:50 "Making the World Supraspinatus Conscious," E. A. CODMAN, M.D., Consulting Surgeon, Massachusetts General Hospital, Boston.

10:20 Discussion.

10:40 INTERMISSION: Inspection of scientific and commercial exhibits.

11:00 "Treatment of Hernia," DAVID H. M. GILLESPIE, M.D., Associate Surgeon, General Surgical Service, Hospital for Ruptured and Crippled; Surgeon, New York Central, and New York, New Haven & Hartford Railroads, New York City.

11:30 Discussion.

11:50 LUNCHEON with Rotary Club. GUEST SPEAKER LOYAL A. SHOUDY, M.D., Medical Director, Bethlehem Steel Corporation, who will talk on "The Doctor Goes to Work in Industry."

THURSDAY AFTERNOON

H. W. LAWRENCE, M.D.,
Chairman of the Session

1:30 "Operative Problems in Rehabilitation of the Injured," HENRY H. KESSLER, M.D., Ph.D., Medical Director of the New Jersey Rehabilitation Commission, Newark.

2:00 Discussion.

2:20 "Physiotherapy Problems in Rehabilitation of the Injured," JOHN S. COULTER, M.D., As-

sociate Professor and in charge of Physical Therapy Departments at Northwestern University Medical School, Passavant, Illinois Central and St. Luke's Hospitals, Chicago.

2:50 Discussion.

3:10 "The Relation of Trauma to Disease and Its Compensability," SIDNEY MCCURDY, M.D., Supervisor Medical Section, The Industrial Commission of Ohio, Columbus.

3:40 Discussion.

Exhibits

Booths 11 and 12

A. S. Aloe Company

1813 Olive St., St. Louis, Mo.

A. S. ALOE Company, world's largest surgical and medical equipment house, will have on exhibit a model first aid set-up for the medium sized industrial plant. A general line of this type of equipment will be shown in their spaces, 11 and 12, together with medical and surgical specialties interesting to the industrial field. The new Gran Portable Stretcher, McKesson resuscitation apparatus, physical therapy equipment and other specialties will be featured. Their representatives will be glad to discuss the Aloe company's planning facilities for industrial first aid departments.

Booths 36 and 37

American Optical Company

10 S. Wabash Ave., Chicago, Ill.

SHOWING a complete line of personal protective equipment including Goggles, Helmets, Respirators, Leggings, Gloves, Aprons, etc. Also scientific apparatus such as Microscopes, Lensometer, Feldman Adaoptometer and other optical precision instruments.

Booth 14

American Sterilizer Co.

Erie, Penna.

AMERICAN STERILIZER Company will display their very latest sterilizers and operating lights for accident and emergency room use in industrial hospitals.

Booth 9

Bell & Howell

1801-05 Larchmont Ave.,
Chicago, Ill.

THE BELL & HOWELL Exhibit will feature the FILMO 70-DA turret model motion picture camera which provides for a variety of lens equipment suitable for every type of critical motion picture work, and which is widely used by medical institutions and individuals for recording cases in natural color motion pictures. Descriptive and operative information on this unusual camera will be given all interested visitors. Complete information will be given regarding the world-wide listing of available medical films as recently brought up-to-date and published by Bell & Howell.

Booth 30

Brooks Appliance Co.

5 N. Wabash Ave., Chicago, Ill.

WE ARE displaying an extensive line of ambulatory and first aid supplies that include only items which have been used by many of the leading and outstanding men in their field.

Peerless Injection Truss—Needles and Syringes

Specially Designed Proctological Instruments

Elastic Adhesive Wound Dressings and Wrappings

Please leave your name at the booth so we may send you our new catalogue and descriptive literature on these special items.

Booth 49

The Cameron Heartometer & Surgical Specialty Company,

666 W. Division St., Chicago, Ill.

THE new Heartometer, a scientific precision instrument for permanent recording of a perfect pulse count and an accurate systolic and diastolic blood pressure reading. It also produces a graph which records the condition of the nerves, valves and muscles of the heart, showing many heart disorders in their earliest and advanced stages. See the new improved Cameron Electro-Diagnostoset, the portable Color-Flash Clinical Camera, the combination Projectoray Diagnostic & Operating Lamp and Projector, the office model Radio-Frequency Cauteradio and the heavy-duty Cauterodynes for all phases of electro-surgery and electro-coagulation.

Booth 27

Burroughs Wellcome & Company

9 & 11 East 41st St., New York, N. Y.

THE greatest single factor in accident compensation costs to Industry is *Infection* following injuries. Many times a trifling injury develops into a "lost-time" compensation case because of infection. Modern ideas in first aid protection aim to remove, or at least reduce, the constantly recurring losses due to infected injuries. Burroughs Wellcome & Co., the internationally known pharmaceutical organization, has developed "Tabloid" First-Aid "Pack-Kits" as a real factor in controlling infection and at the same time increasing the general effectiveness of the first aid kit. The "Pac-Kit" system of first aid is based on the sound idea of a complete dressing for each injury with nothing left over to be returned to the kit as a possible carrier of infection to a subsequent injury. Roller bandages, bulk absorbent cotton and gauze have been entirely eliminated and replaced with complete dressings for individual use. These are so simple in design that even the unskilled can successfully apply them and protect the injury against "further damage" until the patient comes under medical care. "Tabloid" First-Aid "Pac-Kit" materials are prepared under the same precise scientific control as the rarest chemicals and finest pharmaceuticals. Visit the Burroughs Wellcome & Co. Exhibit at Booth No. 27.

Booth 33

The Denver Chemical Mfg. Co.

163 Varick St., New York, N. Y.

IN SPACE No. 33 Antiphlogistine will be exhibited. This product is employed by physicians everywhere in the treatment of inflammatory and congestive conditions. It is kept on hand and used continually in the emergency hospitals and first-aid stations of many of the leading industrial plants in the United States and other countries. Galatest will also be exhibited and demonstrated. This is a new micro-reagent for the instantaneous detection of urine sugar. Galatest has made a profound impression wherever it has been introduced.

Booth 10

Dionol Company

4210 Trumbull Ave., Detroit, Mich.

TIME proven industrial dressings by Dionol.

Plain Dionol ointment. Sometimes called the ideal "skin conditioner" for treating minor burns and other injuries to prevent infection or occupational skin disorders.

Iodized Dionol Ointment. First aid plant dressing for rapid, safe healing without after infection. Iodine burns unknown. Economical and effective. Proven by more than 100,000 industrial dressings.

Analgesic Dionol. A powerful solidified liniment for the promotion of restorative processes in such injuries as sprains of tendons, ligaments and in muscular involvements including the neuralgic myo-

sitis and muscular stiffness due to over exertion.

Booth 22

Doak Company, Inc.

2132 East 9th St., Cleveland, Ohio

VERY interesting clinical reports on the Treatment of Arthritis with Colloidal Sulfur reprinted from medical journals are available. Our dermatological preparations are particularly valuable in the treatment of industrial cases of skin diseases. Doak Dermatological Preparations have been thoroughly tested for the past ten years. Before being placed upon the market they have been clinically tried by some of the leading dermatologists in the United States. The fact will be recognized that the active ingredients in Doak Dermatological Preparations have been used for many decades. In colloidal form and in a suitable base, they conform to modern therapy. We invite you to the Doak Company exhibit.

Booth 48

E & J Company

Drexel Bldg., Philadelphia, Pa.

E & J Company will present a demonstration of the latest development in mechanical apparatus for resuscitation in emergency cases which involve respiratory failure.

Booth 29

Emerson Drug Company

Baltimore, Md.

BROMO - SELTZER is exhibited by the Emerson Drug Company not only to stress its benefits as a prompt relief for headaches and neuralgia, but more particularly to emphasize its usefulness to industrial physicians and surgeons in reducing absenteeism and preventing accidents. Through the prompt relief of headaches, Bromo-Seltzer reduces loss of time among employees to a minimum; furthermore, since the less alert headache sufferer is a potential source of accidents, his prompt relief is a vital necessity in accident and disability prevention.

Booth 46

Ex-Cello Corporation (Pure-Pak)

1200 Oakman Blvd., Detroit, Mich.

PURE-PAK fibre milk bottles. The Pure-Pak container is made of pure, virgin pulp and is carefully safeguarded for sterility in all steps of manufacture. The Pure - Pak method has won the approval of health authorities wherever tested and for years has been the choice of leading dairies from Coast to Coast. Operating in the dairy the Pure-Pak machine forms, sterilizes, parafins, cools, accurately fills, hot-seals, tamper - proof staples and dates Pure-Pak fibre containers in one continuous, automatic operation *without being touched by human hands.* The Pure-Pak machine handles standard quart, pint, and half-pint sizes. And packages fresh milk, cream, sour cream, cottage cheese, orangeade, chocolate milk and other dairy products.

Booth 13

Farnsworth Laboratories

28 E. Jackson Blvd., Chicago, Ill.

THE Farnsworth Laboratories, Exhibit 13, is of special interest to physicians using non-surgical forms of treatment.

The injection of hernia is an increasingly popular subject, and one with which these Laboratories have long been identified.

A local anesthetic built upon a water base is noteworthy for its prolonged action, and wide range of usefulness.

An intravenous treatment for streptococcic and staphylococcic infections is an outstanding product, of particular value in the industrial field, and various other preparations are offered especially designed for ambulatory methods of treatment.

Booth 25

C. B. Fleet Co.

921-27 Commerce St.,
Lynchburg, Va.

PHOSPHO - SODA (Fleet) is a highly concentrated and purified aqueous solution of sodium phosphates. It is non-toxic, rapid but mild in action without irritation of the gastric or intestinal mucosa. Indicated for hepatic dysfunction, and for its eliminating and cleansing action on the upper and lower gut.

Booth 34

Gebauer Chemical Company

9410 St. Catherine Ave.,
Cleveland, Ohio

GEBAUER'S Tannic Acid and Chlorbutanol Solution places in the hands of the physician a spray system of applying an antiseptic dressing of tannic acid. It is a stable solution always ready for immediate use. The Solvent having negligible surface tension, penetrates deeply into minute tissue crevices. Emitted from the Dispenseal bottle with the force of a spray application is more thorough and the Solvent evaporating rapidly leaves a fine residue of evenly applied tannic acid and chlorbutanol. Gebauer's Tannic Acid and Chlorbutanol Solution is indicated in accidental burns, sunburn and minor skin infections and irritations.

Booths 15, 16 and 17

General Electric X-Ray Corporation

2012 Jackson Blvd., Chicago, Ill.

COMPLETELY new in every respect, the G-E Model 39 is a powerful, efficient, single-tube combination X-ray unit. Compact and unusually flexible, with 100-ma. both over and under the table, its range is wide. Its refined, precision control has new conveniences and advantages. The R-39's wide range of service includes fluoroscopy at any angle; vertical, horizontal, and accurate angular radiography with 40-inch tube-Bucky distance. The tube stand is an integral part of the table structure; usual side rail unit is eliminated. Its new, small-size double-focus tube unit provides fine detail over the unit's entire range.

Booth 3

Hanovia Chemical & Mfg. Company

Chestnut St. & N.J.R.R. Ave.,
Newark, N. J.

THE very latest in ultraviolet equipment will be demonstrated, including the outstanding uses of ultraviolet radiation in the fields of science, medicine and public health. Don't fail to see our new line of self-lighting ultraviolet high-pressure mercury arc lamps, Short and Utra Short wave apparatus, Sollux Radiant Heat Lamps and our latest development, quartz ultraviolet lamps for air sanitation — a great scientific achievement to help man fight disease. This apparatus is designed for use in every condition where light and heat therapy have proven effective. Courteous and competent representatives will be on hand to greet you.

Booth 41

Hynson, Westcott & Dunning

Baltimore, Md.

HYNSON, WESTCOTT & DUNNING, Inc., will have an exhibit featuring Mercurochrome and various pharmaceutical specialties of their manufacture. There will also be a display of some of the diagnostic apparatus and ampule solutions which have been developed in cooperation with physicians. As usual, competent representatives of the company will be in attendance to demonstrate the products and to answer questions. Literature and samples will be available to physicians who are not already familiar with products exhibited or who wish to obtain a trial supply.

Booths 1 and 2

Eli Lilly and Company

Indianapolis, Ind.

ELI LILLY and Company, of Indianapolis, feature an exhibit stressing the importance of liver extract in the treatment of pernicious anemia, "Merthiolate" (Sodium Ethyl Mercuri Thiosalicylate, Lilly) in the surgical and germicidal fields, "Sodium Amytal" (Sodium Iso-amyl Ethyl Barbiturate, Lilly) in the field of hypnotics, and Iletin (Insulin, Lilly) in the management of diabetes mellitus. This is the first appearance of the Lilly Re-

search Laboratories at the meetings of the American Association of Industrial Physicians and Surgeons.

Booth 21

Industrial Health Book Company

540 North Michigan Ave., Chicago, Ill.

SAPPINGTON'S "Medicolegal Phases of Occupational Diseases." 2. Gant's "Lead Poisoning." 3. McNally's "Toxicology."

Booth 38

Liebel-Flarsheim

303 W. 3rd St., Cincinnati, Ohio

LIEBEL-FLARSHEIM, Cincinnati, Ohio, will exhibit the well-known L-F Short Wave Generators, as well as the famous Bovie Electro-Surgical Units. In addition, other new and useful physio-therapy apparatus will be shown. A cordial invitation is extended to visit the Liebel-Flarsheim Booth No. 38 to inspect this new apparatus and have it demonstrated to you.

Booth 18

Martindale Electric Co.

Box 617, Edgewater Branch, Cleveland, Ohio

THE Martindale Electric Co. will exhibit Martindale Protective Masks for protection against non-toxic dusts except free silica and asbestos. These respirators permit clear vision and easy speech, weigh less than one-half ounce and are so comfortable that employees welcome them. They are recommended for handling dusty materials like lime, salt, and cement, coal and ash handling; sweeping; buffing; grinding or sanding with artificial abrasives; blowing out motors and other machinery; packing with straw or excelsior; and for general use in textile mills, grain elevators, flour mills, cottonseed oil mills, woodworking plants, leather mills, paper products plants, and similar industries.

Booths 23 and 24

Merck & Co., Inc.

Rahway, N. J.

THE two products of prime interest to industrial physicians and surgeons on display by Merck & Co., Inc., are Mecholyl and Vinethene. Mecholyl, a choline derivative, has proved to be a very effective parasympathetic stimulant. Its effect in increasing the peripheral circulation has been found of value for the relief of pain and other symptoms and signs in arthritis and many painful conditions resulting from injuries, such as sprains, etc. Vinethene (Vinyl Ether for Anesthesia, Merck) is an inhalation anesthetic that produces rapid and easy induction of anesthesia with satisfactory relaxation. Prompt recovery and a minimum of postoperative nausea and vomiting are also features attending its use. Vinethene is a very serviceable anesthetic agent for short major or minor operative procedures. It is also of great value for clinic or office procedures when one desires his patient to be ambulatory following recovery from the anesthetic.

Booth 8

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Booth 47

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8134 McCormick Blvd., Chicago, Ill.

PHYSICIANS and surgeons are cordially invited to visit the new convention display at Booth No. 47 where Petrolagar Labs., Inc. will be represented by Messrs. H. R. Hale and H. W. Scanlon.

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Booth 28

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Booth 35

Physicians' Record Company

161 W. Harrison St., Chicago, Ill.

PHYSICIANS' Record Company, long known as the largest publishers of standardized hospital, medical and dental records, are exhibiting approved forms for Industrial Medicine. Clinical and accounting systems for private practice and for hospitals also are on display. Industrial Physicians and Surgeons are invited to discuss standardized or special forms.

Booths 19 and 20

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Booth 4

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Booths 6 and 7

Westinghouse X-Ray Company, Inc.

Long Island City, N. Y.

WESTINGHOUSE X-Ray will show a new shockproof intermediate-powered machine designed for industrial chest radiography. This will be the main item of the present feature. In addition, there will be shown for the first time two new types of operating room lamps designed for First-Aid Rooms.

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The Industrial Application

IF THERE remains any M.D. who seriously wonders about the fact that industrial medicine has so many close working contacts with other fields of endeavor, he should read the able outline given by DR. T. LYLE HAZLETT, Medical Director of Westinghouse, at the recent meeting of the Council on Medical Education and Hospitals, and published in *J.A.M.A.*, of April 1, 1939, under the title "Fundamentals of Industrial Hygiene."

DR. HAZLETT said: "Industrial hygiene may be defined as the application of medical science to health and its preservation among the working population with the object of establishing the most healthful environment for the worker. Medical service in industry can be divided into three cycles. The first cycle was prior to 1910, when such service was practically unknown. Employees injured while at work were sent to the office of the nearest physician, and the management gave little thought to the surgical care and rehabilitation of injured employees. The second or surgical cycle was predominant from 1910 to 1925, during which years came the development of the group system of production, with an increased concentration of workers. The greatest source of traumatic injuries during these years was in industry, and the result was the enactment of compensation laws. It became apparent in 1913 that many injuries could be prevented, and in that year the National Safety Council came into existence. The third cycle, which began in 1925, can be termed the medical-engineering cycle, since it became evident that many conditions existed in industry that had some bearing on the health of employees. During the last few years more attention has been given to the working environment in its relation to the well-being and conservation of health among workers. Thus there has been opened a new field of preventive medicine, with an industrial application. This requires active participation by physicians working in cooperation with engineers to determine actual measurements of existing conditions so that adequate control measures may be instituted. . . .

"In 1936 an industrial hygiene department was established at the University of Pittsburgh School of Medicine with the object of providing undergraduate and graduate instruction in industrial medical problems and establishing facilities for research on specific industrial problems as they pertain to the health and well-being of workers. In conjunction, an analytic laboratory has been established and hospital facilities are available for the investigation of occupational diseases, together with fellowships for graduates who desire to do such research work on industrial medical problems. An intensified graduate course of one week was offered last year and was well received.

"Perhaps 90% of the medical graduates have in the first years of practice some industrial or insurance contacts which bring them into the compensation field. Consequently there has arisen the necessity for extending the curriculum in our medical schools to provide instruction in industrial hygiene in its application to industrial life and to preventive medicine in industry. This can be done only by making this subject an entity in present day medical education. The part the physician should take and the responsibilities he should assume may be presented under four captions: 1. Safeguarding the health of the industrial workers in relation to their working environment. This includes air hygiene, lighting, noise, fatigue syndrome, sanitation, and placement of workers both from a mental and from a physical standpoint; making periodic examinations, especially of workers in known health hazards, and making definite recommendations to the management for the elimination of any unhealthful practices. 2. Medical care of occupational injuries and diseases and emergency medical care of nonoccupational illnesses occurring while the worker is on duty. 3. Research work. The constant development of new processes, new materials and new combinations of materials and their effects on the human body present to the graduate in medicine today his greatest opportunity in the study of occupational environment and in exploring new frontiers. 4. Health education. Much can be accomplished in an educational manner on the subjects of tuberculosis, heart disease and syphilis, for no group is more easily accessible or more desirous of knowing simple health facts than the industrially employed. . . ."

INDUSTRIAL HEALTH AND DISEASE

The Treatment of Hernia by the Injection Method

ARNOLD S. JACKSON, M.D.,
Madison, Wisconsin

IN 1933 I became interested in the injection treatment of hernia but not until a year later was I sufficiently convinced of the method to try it.* During the interval I had visited the University of Minnesota Medical School and studied the method employed at the hernia clinic conducted by Doctors Bratrud and McKinney. Through reading and investigation, I learned that the idea was not new, but was actually nearly a century old and that a book had been published on the subject as long ago as 1864.

Previous to the work of the Minnesota investigators, there had been reported many cases successfully treated. However, it remained for these Minnesota surgeons in collaboration with Pathologist Bell to establish the scientific merits of this procedure. Yet a large proportion of the medical profession remained skeptical of the idea and it was still associated in their minds with the old quack paraffin method. Actually, there is no similarity whatsoever in the two procedures. One is based on the theory of injecting paraffin to act as a plug as one would cork a bottle. The other, as shown by careful animal experimentation and microscopic demonstration, is based on the idea of injecting a foreign substance into the tissues that will stimulate the fibroblast cells to grow and heal a defect. I believe that this plan of treating hernia will prove to be as efficacious as the injection treatment of varicose veins, hemorrhoids, and hydroceles. There will, of course, be failures and complications as a result of the improper selection of cases by inexperienced persons.

At the Jackson Clinic, my associate, Dr. Luther Holmgren, and I have injected 420 hernias in 358 patients over a period of four years. Although approximately 3,250 injections have been made, but one possible complication has occurred. An elderly gentleman removed his truss a few weeks after treatment was started, allowing his hernia to break through and causing a piece of omentum to become adherent and slough. Following incision and drainage, this rapidly cleared up and his bilateral hernias were cured by completing the injection therapy. The other 3,249 injections have been given with such little disturbance that farmers, blacksmiths, laborers of all types, tend to business and the performance of their routine duties without loss of time. This alone is a matter of tremendous economic saving—when one considers the average cost of a herniotomy to a laboring man, including surgeon's fee, hospital bill, and loss of wages, has been estimated to be $315.00.

The injection treatment of hernia is advantageous from a financial standpoint, but it is even more important from a mortality point of view. Every operation is attended with some risk and unforeseen complication and herniotomy is no exception. From such an excellent institution as the Hospital for Ruptured and Crippled at New York City comes a report which shows 284, or 29% recurrences in 975 operations. The same analysis shows 25 operative deaths and 133 infected wounds with a recurrence developing in approximately one-third. It is likewise significant that 63% of the recurrent cases developed after one year, so that the longer the

* From a paper read at the Utah State Medical Association, Ogden, September 3, 1938; from the Jackson Clinic, Madison, Wisconsin; published in *Rocky Mountain M.J.*, December, 1938.

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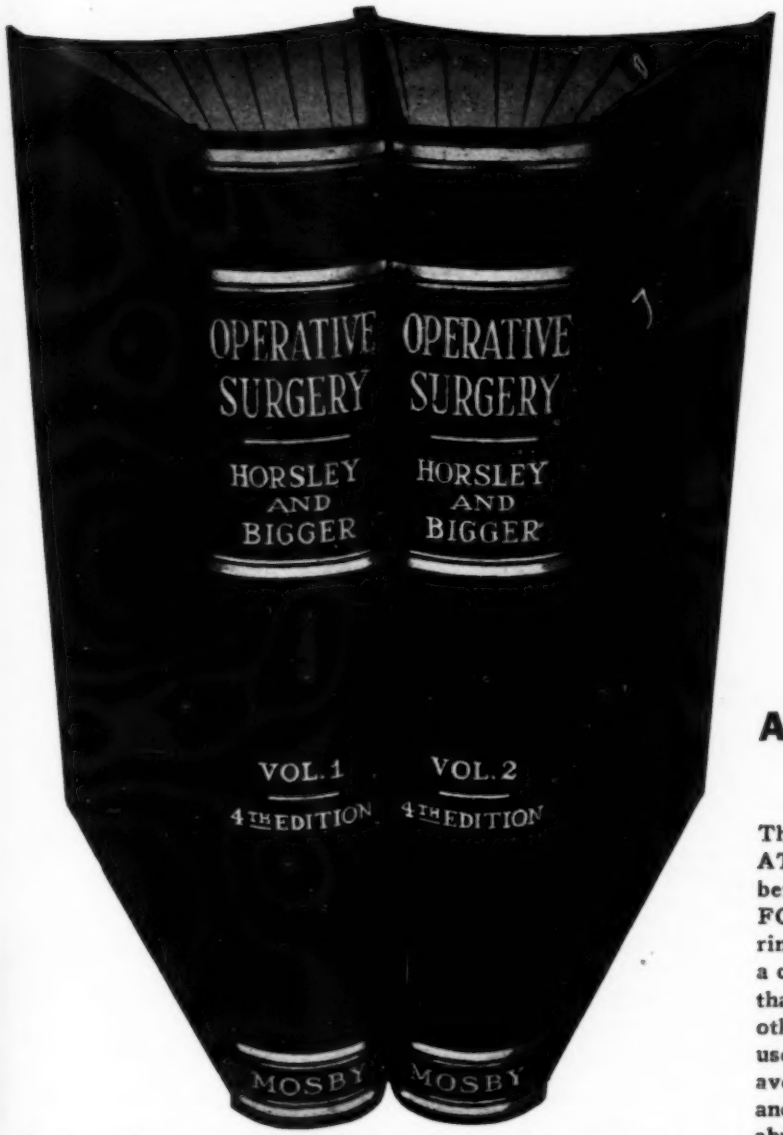

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study is continued the higher is the percentage of recurrence. This excellent fact finding study covered a period of 11 years.

Many surgeons hearing these statistics may feel that their own results are more satisfactory and yet this report comes from an institution of international reputation, one that for many years has been devoted to the treatment of this particular subject. The patient who is unsuccessfully operated upon for hernia frequently fails to return to his surgeon. There are no statistics available on this question of recurrence for the entire country but from my observation of this group of cases, I believe the figure would come closer to 15 than to 10%.

It is evident that surgery has not entirely solved the hernia problem and there is accumulating evidence to show that the injection method may largely supersede the operative treatment. Just as the surgical care of varicose veins, hemorrhoids, hydroceles and ganglia at our clinic has been superseded by injection therapy, so too is this method now used whenever possible in the treatment of most reducible, direct, and indirect inguinal and many femoral and umbilical hernias.

Sufficient time has not elapsed to judge the permanency of results in our series, but at the Minnesota Clinic many hundreds of cases have been followed for several years with a high percentage of cures. We do not feel that until a considerable group of our patients has been observed for at least five years following the last injection that the end results can be accurately stated. We have, however, checked a group of 100 cases after an elapsed period of two years and there is every indication to expect a cure in the great majority. In certain instances where results were unsatisfactory, experience has shown that insufficient injections have been given; that an improperly fitting truss had been worn; that the patient was too obese, or that he failed to cooperate. Careful observation of these points, experience, and the use of improved solutions have given quicker and more satisfactory results.

There is one important difference in recurrence following injection therapy and surgery. In the former the condition may usually be easily corrected by a few more injections. A second herniotomy is usually a tedious and a more difficult procedure than the original one. Many of our most satisfied patients are those who have undergone one or more unsuccessful operations and have then been satisfactorily treated by injection therapy.

It is our feeling that this method should not be attempted without first observing it and receiving instruction from someone who has had considerable experience with this procedure. While the technic is not difficult and may be readily mastered by one familiar with the operative treatment of hernia, serious complications have occurred from its use by inexperienced persons. Consequently, I will not attempt to discuss the technic but will merely summarize the findings in our four-year study.

In a total of 3,250 injections in 359 patients with 420 hernias, a single complication, sloughing of a piece of omentum, due to a failure of the patient to cooperate, is reported. This case was later successfully treated. There have been no cases of infection, impotence, or peritonitis. The injecting solutions are sterile so that with reasonable precaution there is little danger of infection. The hair is not shaved at the site of injection because there is less chance of skin irritation.

Before injecting any solution, one should aspirate to be sure the needle has not penetrated a blood vessel. In six instances this occurred, but the needle was at once withdrawn, reinserted, and following a negative aspiration, the solution was injected. The amount of reaction depends upon the kind of solution, the susceptibility of the patient's tissues to the solution, the site of injection, and the activity of the patient. Proliferol A causes but slight discomfort either at the time of injection or later. Proliferol T may cause a burning sensation and if the patient is active physically, soreness at the site of injection or swelling of the cord for a day or two in some cases. With Sylnasol there is no discomfort at the time of injection, but usually more delayed reaction. About one out of four patients complained of swelling of the cord within a few days after injection. Usually this disappeared in a few days; occasionally it persisted for a week and in such cases injection was not repeated until the swelling had subsided.

In most of our cases, injections were given a week apart until the hernia appeared well closed. The intervals were then prolonged, two or more injections being given a month or two apart. However, in cases that were hospitalized because the size of the hernia was such it could not be held by a truss, injections were given every day or two without causing undue discomfort. While one great advantage of the injection method is that it does not interfere with the patient's occupation, it is desirable for him to refrain from unnecessary activity if a speedy closure is desired.

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